M. H. Gordon

THE

PHYSIOLOGY OF GOUT, RHEUMATISM, AND ARTHRITIS

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PHYSIOLOGY OF GOUT, RHEUMATISM, AND ARTHRITIS

AS A GUIDE TO ACCURATE DIAGNOSIS AND EFFICIENT TREATMENT

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BRISTOL: JOHN WRIGHT AND SONS LTD.

LONDON: SIMPKIN, MARSHALL, HAMILTON, KENT & CO. LTD.

1921

JOHN WRIGHT AND SONS LTD.
PRINTERS, BRISTOL

PREFACE

My medical friends have frequently asked me to explain my 'theories' of Gout, Rheumatism, and Arthritis. I have no theories.

Regarding disorders of the 'arthritic' class not as diseases but as physiological aberrations, I have endeavoured to ascertain the nature of the normal functions and the results which happen when they are imperfectly performed.

By doing so I find myself able to interpret the symptoms which such cases present, and when this has been done the appropriate and efficient remedy for the individual patient suggests itself.

This is only a brief summary of my investigations, but I hope it will prove helpful to the practitioner and enable him to give the 'arthritic' patient a more hopeful outlook than has hitherto been possible.

BATH, PERCY WILDE

October, 1921

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The Physiology of Gout, Rheumatism, and Arthritis.

INTRODUCTION.

THE object of this work is to describe the methods, and line of investigation, which have enabled me to treat successfully a large number of disorders, commonly described, at the present time, as 'arthritis' and 'neuritis', and which are regarded as either intractable or incurable.

It may be expected that I shall begin with the announcement that I have discovered some drug which, if taken continuously, will restore the joints and the tissues to their normal condition; or, that I have invented some infallible system of diet, which will prevent the poisons, which cause such destructive changes in the joints, from being manufactured in the body; or, failing this, that I have found a vaccine fatal to some bacillus which I have proved to be the source of all the mischief.

I have done none of these things; thirty years' experience in the daily treatment of such cases, and much experimental work, has not encouraged me to hope for a cure from any of these methods. It is true that drugs have been found which, because they relieve some of the symptoms met with in such cases, have raised hopes that a true specific would, at length, be found;

but centuries have passed and the expectation has not been realized.

The chemical school, as a result of numberless experiments, concluded that an excess of nitrogen introduced into the body was a serious cause of joint trouble, and that, by severely limiting the nitrogenous foods, great results might be expected. This view has been very generally adopted, with the result that for many years 'diet' has taken the place of the 'weather' as a subject of conversation at the public dinner-table, and abstinence from certain foods has been raised to the level of a virtue by some people. But the number of those who suffer from 'arthritis' has not diminished. During the war we took thousands of men, not too well fed, and of no great physique, and we crammed them with nitrogenous foods; but instead of developing joint troubles they made bone and muscle, and acquired powers of endurance which enabled them to withstand the severe physical strain to which they were exposed.

In the course of my investigations, I had to examine the two processes used by chemists to discover the influence of foods on the excretion of uric acid. I found that in both methods a very remarkable mistake had been made, which I shall describe in the chapter on uric acid. I found that in the solution used for estimating the quantity of uric acid there was none present, and the only nitrogenous product in the solution was added by the chemist in the course of his experiment!

The bacteriological theory which is now widely accepted makes a greater appeal to the imagination, because we know that certain bacilli are responsible for serious mischief to joints. Thus, we know that the gonococcus bacillus is capable of producing severe

arthritic symptoms, and that these may continue for an indefinite period. To a lesser extent this is true of other bacilli, such as the staphylococcus and streptococcus.

But before we can accept the view that these bacilli are the primary cause of the joint trouble, we have to explain how it happens that a hundred people can have their tissues invaded by the gonococcus bacillus without developing the smallest symptom of 'arthritis'; and, when we come to other bacilli, they may be found in the tissues of hundreds of people before we come upon a case where arthritis and the bacillus are co-existent.

We can only explain this by saying that there must exist some prior condition of the tissues which weakens their resistance to the attack of the bacillus. If this is true, the destruction of the bacillus will only relieve the symptoms it causes, and not the condition of the tissues upon which the disorder depends. This conclusion is justified theoretically, and also by the results of efforts to cure the disorder by the use of vaccines.

Thus, in chronic rheumatism we find that in the large majority of cases the cause is prolonged or repeated exposure to cold.

We can demonstrate that this has caused an impairment of certain normal functions of the body; that this, in turn, has caused changes in the normal metabolic processes of the body. We find that this condition may continue for a long period before any symptom is recognized to which the word 'rheumatic' can be applied. But during this time there is a gradual alteration taking place in the fibrous and cartilaginous tissues, and it is only when these have made considerable advances that the early symptoms of 'rheumatism' are noted. It may happen that during the whole course of the disorder no symptom of fever or inflammation

appears. The clinical history is the antithesis of the invasion of the tissues by a bacillus, or even the administration of vaccines, as both are associated with a reaction of the organism and an elevation of the temperature of the body.

To understand how some simple functional impairment can result in serious changes in the tissues, it is necessary to have some knowledge of the normal metabolic processes and the conditions which govern their mechanism. In studying these problems, I found very little help from either physiological text-books or medical literature.

Sir Michael Foster, in his great work on physiology, concludes his chapter on metabolism with the remark that " the whole story of proteid metabolism consists, at present, mostly of guesses and gaps ". As far as I can discover, little progress has been made since this work was written.

I have had to ascertain the facts by personal observation and experiment. I first examined the human excretions with the microscope, under varied conditions ; and then, in order to find out the chemical composition of the crystals observed, I used the method of synthesis instead of chemical analysis. That is to say, I reproduced these crystals artificially. I found that this not only gave information unattainable by chemical analyses, but also prevented me from falling into some mistakes made by the early observers and which have been repeated by all subsequent writers. In fact, the whole uric-acid theory has been based on these mistakes. My difficulties throughout the investigation have been less with the normal processes of nature and the changes which produce these disorders, than with the statements and theories which have been so long and so universally

accepted as basic truths that it appears almost impious to reject them.

But when I had done so, the obscurity, which has always clouded the clinical interpretation of the phenomena presented, disappeared, and it became possible to find a physiological explanation of every symptom met with in daily practice. I found that the processes of metabolism studied by the method I adopted became more intelligible and less intricate than they appeared from the purely chemical standpoint, and what struck me most was the evidence of purpose and design in all the actions and reactions observed. I found that there were no 'waste products', that each had some definite duty to perform necessary to the well-being of the organism. Thus urea, when not performing important functions, was a wholly innocent body, and even uric acid was created for a beneficent purpose.

Metabolism represents unceasing chemical change: but what surprised me was how little these changes were influenced, under normal conditions, by any agent we administer either as drug or a food. The most important causes of chemical change are purely physical, such as cold, heat, and exercise; and because it is a part of the scheme of nature that this should be so, these changes largely take place in the great lymph-space which lies immediately beneath the skin throughout the whole surface of the body.

We all know how much the symptoms of the rheumatic patient are influenced by heat and cold, rest and motion; but we do not always realize that these symptoms are the result of definite chemical changes taking place in the tissues.

The importance of this large lymph-space is very

great, and a knowledge of the physiological and chemical processes taking place in it are necessary before we can understand the nature of gout, rheumatism, or arthritis; but still more important is a knowledge of the special organs designed to eliminate the metabolic products of our tissues. The skin has been described as regards its structure, its capacity to regulate the temperature of the body by the contraction and dilatation of its blood-vessels, and its two *secretions*, one of sebaceous matter and the other of sweat, but as the great excretory organ of the products of tissue metabolism it is practically unrecognized.

It is because this is so, that physiologists, looking for the results of muscular metabolism in the excretion of the kidney and failing to find it, after vigorous exercise had been taken, have been driven to invent many speculative theories.

We have patients becoming hopeless cripples as a result of an impairment of its functions. We may have direct clinical evidence that a chill was the primary cause, and we try drugs, diet, and vaccines; and because no satisfactory results follow our efforts, we pronounce the disorder incurable.

I do not wish to convey the idea that the skin is a mere outlet for the excretion, and that its channels may become clogged, and all that is necessary is to 'open the pores of the skin'. The functions of the skin include the oxidation of the waste products as well as their excretion, and it is provided with all the machinery for this purpose. *We may obtain excretion from the skin without restoring its functions.*

The skin does not merely control the temperature of the body, but is itself a source of heat. For this reason functional inactivity of the skin causes the

temperature to become subnormal, and this occurs in men otherwise in robust health.

But while a restoration of the function of the skin is a primary and essential condition in the cure of these cases, this will not in itself cure the secondary changes which have taken place in the tissues. This involves the study of another series of factors, which we are helped in understanding by observing the processes which nature adopts to overcome them.

I could describe a very large number of cases diagnosed as 'rheumatoid arthritis' and given a hopeless prognosis, who have made a complete recovery. But if I gave details of the treatment of each case, I should have done very little to advance our knowledge of the subject. The term 'rheumatoid arthritis', as commonly used, conveys no information as to the cause of the disease, the constitutional condition of the patient, or the condition of the affected joints. The treatment indicated in one case, called by this name, would be useless or of little advantage in another case. When I read theories as to the causes of 'rheumatoid arthritis', or statements respecting remedies found useful in its treatment, I wonder which particular class of cases the writer is thinking about; all the conditions differ so widely in cases called by this name that there is no scope for generalization.

We are chiefly concerned in such cases with the restoration of the functions of the joints. In every case of joint disease there is only one line of treatment indicated by the condition of the joint, and unless this is followed the result will be failure. If we regard any chronic form of joint trouble merely as a symptom of a disease, and try to treat it by finding a remedy for the disease, we shall be almost certain to neglect the obvious

indications for treatment; thus, the patient may be allowed to walk about with a knee-joint in an inflamed condition while we are treating him for the theoretical cause of the disease. We cannot say that any particular treatment is indicated for different varieties of joint trouble, because the treatment required for a joint at one period would do injury to the same joint at another.

Neither can we say that in any particular case the treatment required by one joint or more would be equally valuable for all the joints in the same patient; on the contrary, it might do considerable harm.

The proper treatment in any case can only be ascertained by careful physical examination of the joint, so as to ascertain the exact condition of its tissues in regard to the processes taking place in them. Every case thus becomes a physiological problem, and is really more interesting than the majority of cases with which the physician has to deal.

It is to help the practitioner to know the exact physiological cause of the symptoms presented, so that he may be able to apply the indicated treatment, that I am writing this book.

CHAPTER I.

SOME CLINICAL FACTS.

It is a natural instinct, when we see a patient suffering the pains of acute gout or rheumatism, to seek some remedy which will quickly relieve his sufferings. To some extent we have it in our power to do so. We may give colchicum in sufficiently large doses to relieve the pain of acute gout and shorten the attack. But clinical experience teaches us that, if we treat gout in this way, the attacks become more frequent and more severe, and there is a concomitant failure in health. The abortive treatment of acute rheumatism by large doses of salicylates is followed by frequent relapses and prolonged convalescence; and heart disease occurs in about 70 per cent of the cases treated.

These results happen, not because there is anything peculiar or erratic in the nature of these diseases, but because we have used language which conveys a false conception. If I state that neither rheumatism nor gout, regarded *as diseases*, is accompanied by pain, I shall not be understood, because we have become so habituated to associating the symptoms with the disease. But we shall never attain success in the treatment of such cases until we recognize the difference between the symptom and the disease. The word 'disease' can only be properly applied to any agent or condition which affects the functional activity or vitality of any part of the organism. The 'symptom' is produced by the reaction of the organism against this

agent, or the failure of that reaction (secondary symptom). The active symptom is thus a force moving in an opposite direction to the disease.

The pain, swelling of joints, and fever we meet with in cases of gout and rheumatism do not represent the disease, but the reaction of the organism against it. The disease itself has none of these symptoms, but one or all may be produced by the effort of the organism to cure the disease. Therapeutic measures have failed in these disorders because the misuse of language has created false ideals.

The cause of the disorder may exist in the organism for months or years without producing symptoms, but if it does they may not be the symptoms usually associated with the words gout and rheumatism. Thus many cases of rheumatic fever escape recognition because the joint symptoms usually associated with the disease are absent.

The efforts of the organism, represented by the symptoms, are very frequently incomplete because the power of vital resistance is limited; but if we mistake these efforts for the disease and endeavour to suppress them, we range ourselves on the side of the disease and allow it to win the battle. The organism may make fresh efforts, after a time, when the forces we have brought to bear against its resistance have been removed, and then we call it 'a relapse'; or we may so greatly diminish the power of vital reaction that the disease becomes symptomless, whilst it is working that mischief on the heart or kidneys which the organism and the protective powers of nature were trying to avert.

Thus, in the eighteenth century, a foreign Convent prepared a powder, of which one dose taken every

night for a year quite arrested the attacks of gout in those suffering from the disease. The Duke of Portland purchased the recipe for a large sum of money, and presented it to suffering humanity. But it was found that the freedom from the attacks of gout usually resulted in death from disease of the kidney. Dr. Falconer, of Bath, wrote describing his experiences with this remedy in the *Medical Journal* towards the end of the same century, and as a result its use was given up by British practitioners and has long since been forgotten. But its employment has been revived, and I have personally seen two deaths from nephritis as a result of its use. The rheumatic-fever patient may make a brilliant recovery from the acute attack as a result of large doses of the salicylates, subsequently to lead an invalid life, and finally die of heart disease.

Not only is it important to recognize symptoms and diseases as forces moving in opposite directions, but it is necessary to remember that a similar relation exists in the medicinal and physical agents we employ in treatment. This is obscured in our works on therapeutics, because writers describe the symptoms produced by medicinal agents as the 'action' of the drug.

If we except a limited number of drugs which enter into chemical combination with protoplasm, and may therefore be regarded as chemically active, we may say that all drugs act as physical stimuli, and the symptoms they produce are due to the reaction of the organism and represent, therefore, a force moving in an opposite direction to the drug. The 'action' of a drug is governed by physical laws, and this action will increase in intensity as the dose is augmented. The

'effects' produced by drugs are governed by the physiological laws of stimulation and exhaustion. Any agent which can stimulate an organic element has also the power to exhaust it, so that a physical agent setting up an '*action*' which is a force moving in one direction with increasing intensity, can produce *effects* which are exactly the reverse of one another.

It is a fact that light stimulates the sense of sight, and sound that of hearing; but it is not a truth unless we also state that excess of the *action* of light has the *effect* of blinding, and that excess of sound is deafening.

It is important that we should keep these facts before us. Thus, when we use heat to stimulate the functions of the skin, we must not forget that excessive heat may seriously impair its functional capacity. The galvanic current has an action which stimulates the activity of the nervous system, but by no agent can we so quickly cause its exhaustion.

Clinical experience helps us to avoid some of the evils which would result from failure to recognize these elementary facts. We do not try to relieve the heat and swelling of a joint afflicted with acute gout by plunging it into cold water; we have learned that such treatment would be disastrous. On the contrary, we wrap up the affected joint and increase the heat and the swelling; and in doing so we are using agents which are moving in the same direction as the reaction of the organism, and therefore we know that a right course is being taken.

But the conditions we meet with in everyday practice are so varied that we cannot depend upon our own clinical experience or that of others; for guidance we must have some definite principles to enable us to elucidate the problems presented and enable us to take

the right course. Neither the recognized routine treatment, nor the results of the clinical experience of others, can be accepted as a safe guide. The gap in the hedge may appear the best way out of the difficulty; but even if this gap has been used by generations of others until it has become a well-trodden road, unless it leads us to our goal we must fall back on first principles to help us to seek another way.

Thus, the symptoms of acute gout have been known and recorded since the beginning of history. The patient is suddenly seized with violent pain and acute inflammation in one or more joints which were apparently healthy at the moment of the attack. The ancients took the view that this was due to some morbid matter in the blood which was suddenly instilled into the joint. Rodulfe in the thirteenth century crystallized this theory by giving this disorder the name 'gout', from the French 'goutte', a drop, to signify that the poison entered the joint drop by drop. This view has been so universally adopted that we can express the attack in practically any language by using the word in that language which is equivalent to 'a drop'.

This view embodies two clinical conceptions: first, that a healthy joint is liable to be attacked with acute gout; secondly, that the poison which causes the inflammation is derived from the blood. The whole literature of gout has been built upon this foundation. The variations which occur in the writings of successive centuries have been as to the exact nature of the poison which exists in the blood, and the best methods of preventing its collection or of eliminating it when collected. What evidence have we that these clinical observations are true?

The first conception rests entirely upon the evidence

of the patient. For two thousand years he has told the physician that his joint was quite healthy at the moment of the attack, and for two thousand years the physician has believed him.

Is the patient a credible witness? My first investigations into the nature of gout, undertaken about thirty years ago, had reference to this point. I examined the joints of a very large number of persons, regardless of age and sex, who had no symptoms of gout or any trouble with the joints. I was surprised at the frequency with which I discovered that fine crepitation which indicates the presence of foreign matter in or around the joints.

The usual term for these tiny concretions is 'urates'; but they were formerly called 'lithates', and I shall use the latter word, not because I am old-fashioned, but because it does not commit me to any theory of the chemical nature of these concretions.

The patients who had 'lithates' in their joints were for the most part wholly unconscious of the fact. Therefore the statement of a patient that his joint was perfectly healthy at the moment of the attack is valueless. I made careful note of the position and size of these lithates, and I had the opportunity in many cases of watching their history over a long term of years. In some cases the lithates spontaneously disappeared. I have seen cases where a few days' rest in bed would produce a crop of lithates, but these would disappear directly the patient resumed active exercise. In many cases these lithates would remain for many years and give no indication of their presence, but in some cases they would increase in size and give audible evidence of their existence without causing pain. In other cases a certain amount of

irritation was set up during movement by their mechanical effects.

I became accustomed to regard all persons who had this tendency to deposit lithates in their joints as having the 'lithic diathesis'. I took particular note of the physiological conditions and symptoms presented by these patients. I called all joints where lithates were present 'lithic joints', and I use the term even when pain exists, if the pain is due to purely mechanical irritation.

But in some of these patients the lithates underwent chemical change; there was pain in or around the joint, the severity of which depended upon the degree of chemical activity which was taking place. When it was very rapid and complete, the patient exhibited the symptoms of acute gout. I call all these manifestations of inflammatory changes produced by the decomposition of the lithates 'lithitis', and use the adjectives acute, subacute, and chronic to qualify it.

I use these names because it is very difficult to describe the symptoms we constantly meet with without a better nomenclature than we possess, one which often commits us to false theories of the nature of the disorder. I can say as the result of my observations that it is absolutely impossible for any healthy joint to be attacked with gout. The lithates, the decomposition of which cause the attack, have existed for months or years before.

I have read statements made by writers on gout that the deposits are due to the attack of gout and increase with the recurrence of the attacks. This statement is based upon defective observation. In every case the lithates present are greatly reduced in size by the attack, but in no case, as far as I have had experience, do the

'tophi' wholly disappear as a result thereof. This point is of clinical and physiological interest, and I will explain the reason for it later.

At this stage, I can reasonably expect that others will examine the joints of healthy persons to see whether lithates can be found as frequently as I have described. It is necessary, therefore, to describe what I mean by the examination of a joint. We may inspect a joint, palpate it, or have an *x*-ray photograph taken of it, and we may obtain much valuable information as a result, but we have not placed ourselves in a position to give an accurate diagnosis of its condition until we have examined it during the performance of its functions. I could describe large numbers of cases where very serious errors of diagnosis have been made because this fact was not recognized.

If the palm of the left hand or the palmar surface of the fingers, according to the size of the joint, is pressed firmly on a joint, and then with the right hand the joint is put through the extreme movements of which it is capable, the smallest deviation from the normal condition is at once communicated to the hand, and by a little manipulation the exact site can be located. The fine or coarse crepitation of lithates is unmistakable, and conveys an accurate idea of their size, while the soft creak which results from destruction of tissue is very evident. The sounds can be accentuated by causing the patient to make the movement of the joint while resistance is offered to it. The hand soon becomes educated to the smallest deviation from the normal, and it becomes unnecessary to remove the patient's clothes when examining a joint, unless other conditions render this essential.

We now come to the second proposition, the relation

of the chemical constituents of the blood to the attack of gout.

If we agree that the lithates exist in the joint for months or years before the attack, is the blood responsible for their deposition? The universal opinion of the profession is in the affirmative. Personally, I think no single practitioner has a right to hold that view unless he is in a position to explain how a chemical body so highly soluble as to circulate in the blood can suddenly become an insoluble body when it reaches certain tissues. If he holds this view, he ought to be able to explain further how the insoluble body takes such a hold on the tissues that all the great forces brought to bear upon it by the movements of the joints fail to dislodge it.

This subject is so important that I shall devote a special chapter to its consideration, but I mention it here in order to show the difficulties in which we are placed if we adopt theories which have been formulated on the 'gap and guess' principle. Clinical evidence is in itself often misleading, and we have no room for theories which will not bear investigation.

Leaving the source of the lithates for the present, we have to consider whether the blood is directly responsible for the chemical change taking place in the lithates which produces the pain of gout. Here clinical evidence may lead us to answer the question in the affirmative. Every practitioner can record cases where the pains of gout have been induced by eating fruit, or drinking port wine or beer, or by too much 'red' meat, or the use of sugar. This creates the impression that they alter the state of the blood, and the lithate undergoes decomposition in consequence. But large numbers of lithic patients can take any or all of these things, not only without

suffering, but to their great advantage. We are not only confronted with this problem, but with one that is much more difficult to explain. I make a rule, when a patient comes to me with lithitis in one or more joints, to examine all the joints of the limbs, and almost invariably I find that the lithates are not peculiar to the joint affected, but exist, perhaps in greater number, in other joints, although they give no trouble and the patient may be quite unaware of their existence. If it is the state of the blood which has set up the decomposition of the lithates in one joint, why does the same blood have no effect upon the lithates in the other joints?

This is not a momentary matter; the affected joint may continue in a state of irritation for many months, and during the whole of that period the lithates in the other joints may give no indication of their presence. It may happen, twelve months after the affected joint has become quite well, that these other lithates commence to undergo decomposition, and the symptomless joint becomes the site of 'chronic gout'.

These difficulties have led to the idea that there is something mysterious and difficult to understand about gout; but in reality it is the theoretical conclusions which prevent us from having a clear conception of the nature of the physiological deficiencies which give rise to the symptoms.

In the treatment of all disorders of the gouty and rheumatic class it is absolutely necessary that we should know, not only the physiological causes which produce the disorders, but also the exact condition of the patient at the moment of examination. We can ascertain these facts by knowing what happens to the waste products of the body after they are formed, and how they are

excreted. It will be found that in the study of metabolism this subject becomes less difficult and complex the nearer we approach the truth. We can by certain tests ascertain the exact position of the patient in regard to these processes at any moment. But nothing will take the place of accurate physical diagnosis, because we are never treating an abstract name, but some definite deficiency or series of deficiencies in the individual patient, and these will not be the same at any two particular periods of treatment. Thus, absolute rest, and vigorous exercise, may be essential conditions at different stages of the same disorder, and when to order the one or the other can only be ascertained by careful physical diagnosis. The symptoms in any two cases are never precisely alike, because the possible combinations of conditions are incalculable. For this reason there can be no routine treatment suitable for even a large proportion of cases.

There is really no class of cases which is so interesting to study or so much worth it, because in the past they have been regarded as incurable.

As the urate is credited with being the source of most of the troubles of gout, I will give it the first consideration.

CHAPTER II.

THE GENESIS OF THE URATE.

Urea has its origin in the dead cell. It is the protoplasm of the dead cells taken as food which constitutes the largest source of its supply, but we are chiefly concerned here with that which is derived from the dead cells of our own tissues.

If we followed the ordinary course, and considered the dead cell after it had been resolved into its chemical constituents and these had undergone transformation, we should evade one of the greatest physiological problems with which nature is concerned, and fail to understand many important clinical facts. The dead cell is an insoluble particle of organic matter, and it occurs in aggregations large enough to block the capillaries if they were allowed to escape into the circulation. The dead cell, because it is dead, is liable to undergo putrefaction.

Here are two difficulties which nature has to meet in the normal disposal of the dead cell, in every tissue except that of the skin. In the skin it does not matter; the dead cell has only to dry up and drop off. But even this description as regards the skin is not correct. If we bathe the skin in hot water, and apply soap, and rub it dry with a towel, and repeat the process day by day, we have not removed the dead cells. We can prove this by taking a vapour bath and producing the act of sweating. If we now rub the surface of the skin, dead cells will come away in large flakes, which

roll up as we pass the hand over the skin. This shows that the dead cell is not only firmly adherent to the living cell, but is also firmly joined at its edges.

It is a definite physiological law that the dead cell shall remain in cohesion to the living cell until it has undergone such chemical change as will render it fit for removal. The delay which takes place in the epithelial cells of the skin does not appear as urgently necessary as it must be for the cells of the other tissues. We have a right to infer that what we know actually takes place in the skin must take place in the dead cells of the other tissues. We may go further and say that the dead cell of the tissues *must* become soluble before it is removed from the living cell.

If this conclusion is correct, and we accept the teaching that urea is formed in the liver, we must imagine some other soluble nitrogenous body to be formed before it can be carried to the liver. As urea is an ultimate product of decomposition, I have never understood the reason for supposing that some special organ was responsible for its manufacture. The dead leaf drops from the tree, falls to the ground, and becomes resolved into its ultimate constituents without the aid of any special organ to perform the work; why should it be different in the case of the dead cell? It has been suggested that carbonate of ammonia is the soluble nitrogenous body and that this is the 'precursor of urea'.

But while urea is constantly being converted into ammonia in the body, there is no evidence to show that the reverse process takes place. The conversion of ammonia into urea is not an easy process for the chemist to accomplish. Why should nature adopt the extremely clumsy method of making carbonate of

ammonia and then having it conveyed to the liver to be converted into urea?

In order to study the physical and chemical conditions of the dead cell, I procured some of the tendons of an ox, directly after it was killed. I selected the ox because it leads a sedentary life, and the metabolic changes are likely to be slower. It was necessary to make my examination immediately after death, as chemical changes in the tissues rapidly follow. I used the tendons because their smooth, white, fibrous tissue has no interstices in which adventitious matter could rest.

I placed the tendons in water at a temperature of 100° F. for half an hour. I then decanted the fluid, and found it was slightly opaque, with a faint yellow tinge. Examination under the microscope revealed a large number of cells showing different degrees of disintegration. These were dead cells which had been attached to the living tissue, although the smooth glistening appearance of the tendon gave no indication of their presence.

As I removed these cells by simply immersing the tendons in warm water, it might be reasonably asked why they were not removed by the lymph at the same temperature during life. It must be remembered that a change takes place at the moment of death which loosens the cohesion of the dead cell to the living tissue. I shall consider the nature of this change later. This experiment conclusively proves that the dead cell remains in contact with the living tissue until its metabolism is complete.

I tested the solution for ammonia, and found it absent, because the animal was freshly killed, and on testing for urea with hydrobromite solution I found it present in large quantities.

I think this experiment demonstrates the fact that urea is formed in the tissues as a normal product of metabolism, and is not specially manufactured by the liver or any other organ.

I found in my solution urea, lactic acid, and chloride of sodium, in addition to the dead cells. Now as lactic acid and urea have a great affinity for one another, we might suppose that they existed in combination as lactate of urea. But I found no evidence of either lactate of urea or lactate of ammonia in my solution. The investigation of the reason for this gave me some unexpected information which throws a great light upon the problem of metabolism.

Urea has the formula $CO(NH_2)_2$, and is a very unstable body. It will be seen that it only requires an atom of oxygen to convert the CO into carbonic acid gas, and only two atoms of hydrogen to convert the $(NH_2)_2$ into ammonia. Thus a single molecule of water, if its atoms were free, would convert urea into carbonate of ammonia.

These facts convey an idea of the chemical nature of urea, but we are concerned here with the great affinity which urea has for acids. It forms many salts which are fairly stable, but which, when decomposed, yield ammonia ; but in the human body the acid it combines with is lactic acid, and the salt formed is so unstable that we never find it in the excretions. It changes rapidly into lactate of ammonia. Lactate of urea forms crystals which resemble long slips of clear glass. I tried to photograph some which I made, but during the process they became decomposed, and the photograph (*Plate I, Fig.* 1) is only interesting as showing the decomposition taking place.

Urea forms long silky needles as we find it outside

the body (*Plate I, Fig.* 2), but under no circumstances do we find a crystal of urea in the lymph or the excretions of the body. These facts bring us to a very important source of confusion in the whole subject of metabolism.

The endeavour has been made to express physiological truths in the language of chemistry, and the disadvantage of doing so is shown by the first solution we come in contact with. When I stated that I found urea in my solution, I used chemical language. Urea is there because it answers to chemical tests; but urea as a definite crystalline body having certain affinities was not there, otherwise it would have combined with lactic acid and formed lactate of urea. The chemist takes no account of these facts. When he analyzes a solution of Epsom salts, he tells us that it contains so much sulphuric acid and so much magnesium; but as a matter of fact the sulphuric acid has lost its hydrogen and ceased to be an acid, and we can produce none of the physiological effects of sulphuric acid with sulphate of magnesium.

We can understand urea existing in the blood and the lymph, because it is stable in an alkaline medium; but, How can it pass through the kidney and find itself in a highly acid solution and not become converted into ammonia? I do not think that anyone has ever asked the question or attempted to solve it.

The chemist proves that the urine contains a large amount of urea, and Sir Michael Foster was led to believe that urea had an independent existence in the urine, and states so.* But no crystal of urea is ever found in the urine, and it would be impossible for it

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* *Text-book of Physiology*, p. 649.

PLATE I.

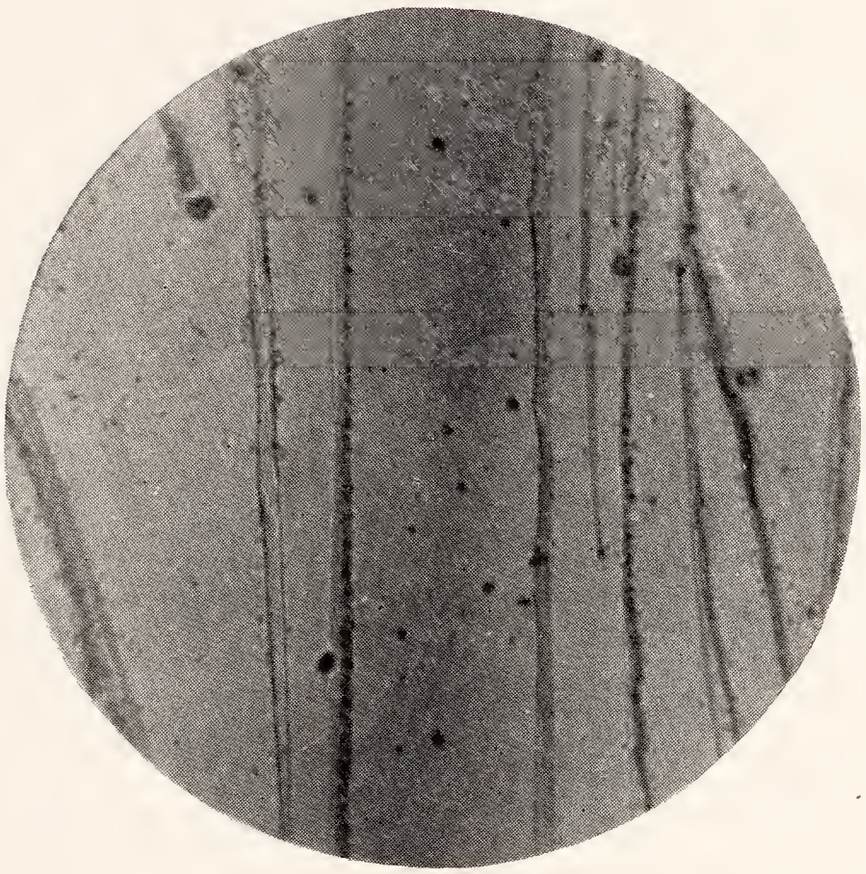

Fig. 1.—Transformation of Lactate of Urea into Lactate of Ammonia.

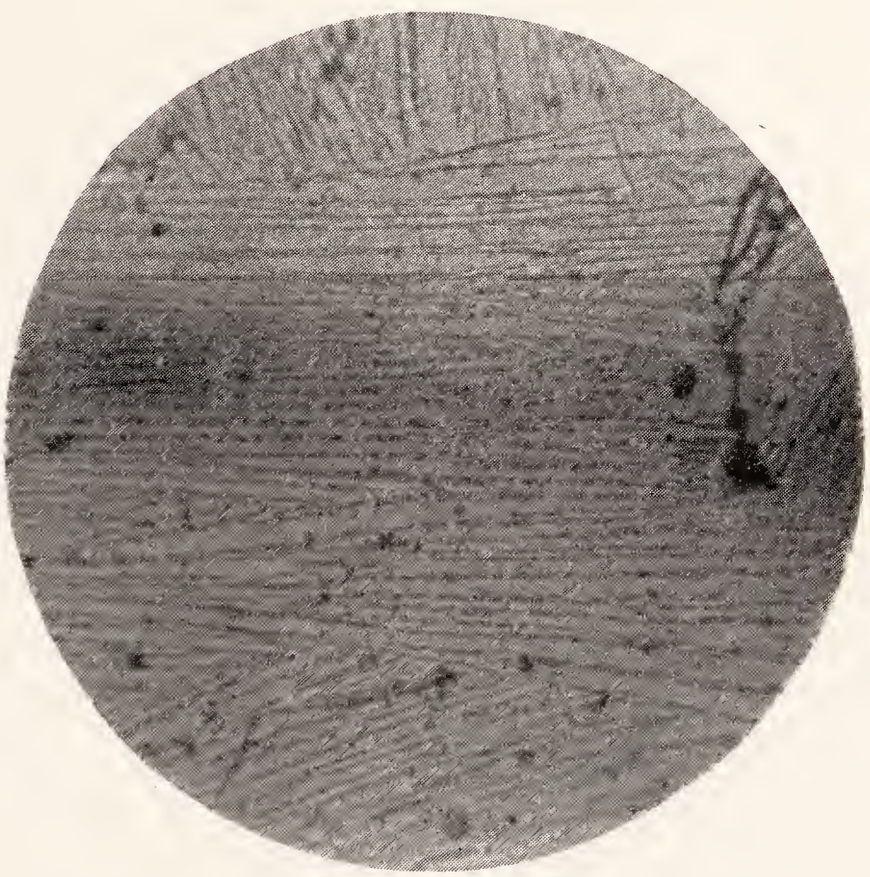

Fig. 2.—Urea.

Facing page 24.

to exist in the urine without transformation into ammonia. In the same way urea must meet free lactic acid in the lymph. Why does it not combine and become converted into ammonia?

This points to the fact that urea must enter into some chemical combination which enables it to resist the action of acids, and a clear understanding on this point is absolutely necessary to a knowledge of the metabolic processes. Now urea exists in such large quantities that we pass in the urine about 512 grains every day. Therefore any body which combines with urea must also exist in large quantities.

This directs our attention to chloride of sodium, which occurs in such abundance in the fluids of the body. Chloride of sodium is a very stable salt, but viewed in the crystalline form has a remarkable tendency to change its appearance. Various forms of chloride of sodium are illustrated in the text-books. From the physiological point of view we must regard every change in the external form of a crystal as due to some alteration in its chemical composition, and therefore we must look upon the various forms in which chloride of sodium disguises itself with some suspicion. It appeared to me advisable to see what influence urea had on the formation of the chloride of sodium crystals contained in the same solution.

I made a solution of sodium chloride (4 gr. to the ounce) in distilled water. To this I added ½ gr. of pure urea. This was placed in a water-bath at 100° F. for one hour. A gas was evolved, showing that chemical change was taking place.

A drop of this solution, evaporated and examined under the microscope, showed that the normal square crystals of chloride of sodium had disappeared, and that

crystals of the 'dagger' type had taken their place. As this type of crystal is not uncommon, it is necessary to observe that those formed by the combination of urea and chloride of sodium have a smooth edge and a central rib, resembling the petal of a flower; and many of them have these of equal length, so as to resemble a flower rather than a dagger. These crystals represent a definite chemical body composed of one part of urea to seven parts of chloride of sodium. It is not a double salt, because urea is not a salt; the sodium chloride has not been decomposed, but as a gas has been evolved

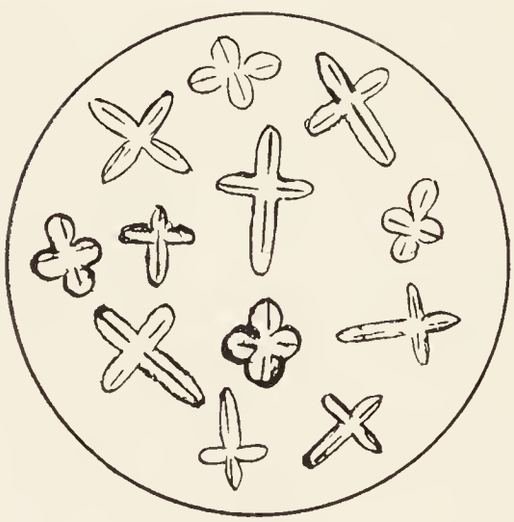

Fig. 3.

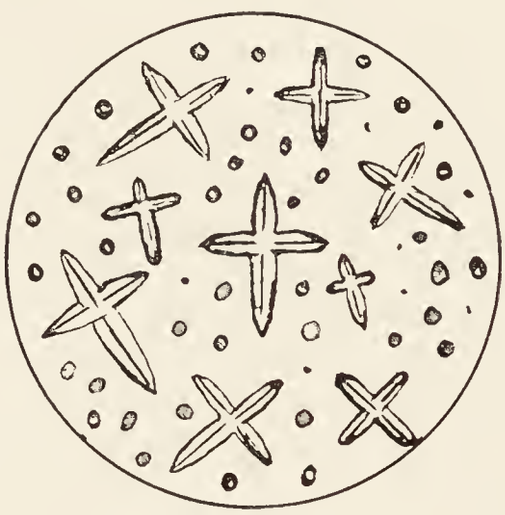

Fig. 4.

during the combination, it seems probable that the urea has given up an atom of hydrogen in the process. This point I have not determined (*Fig.* 3).

I repeated this experiment, using urea 2 parts to sodium chloride 4 parts. I again found the dagger-shaped crystals and their modification, but small round crystals appeared dotted about between the larger crystals (*Fig.* 4). I next repeated the experiment, using urea 2 parts and sodium chloride 1 part. The result was the dagger crystals entirely disappeared and the whole field was covered with the small round crystals

PLATE II.

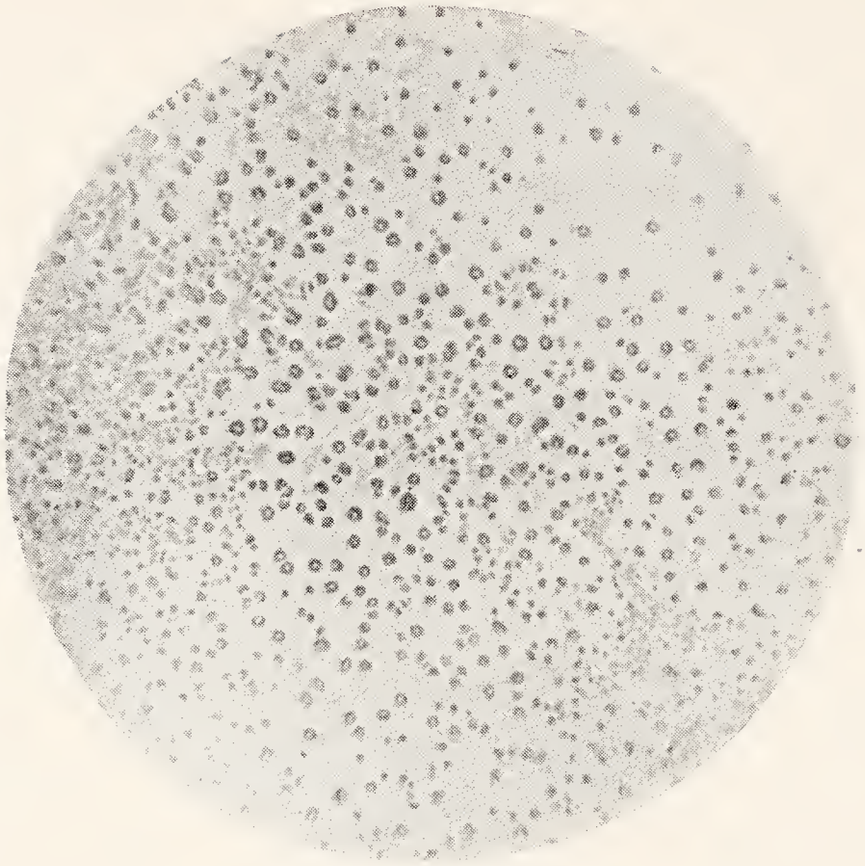

Fig. 5.

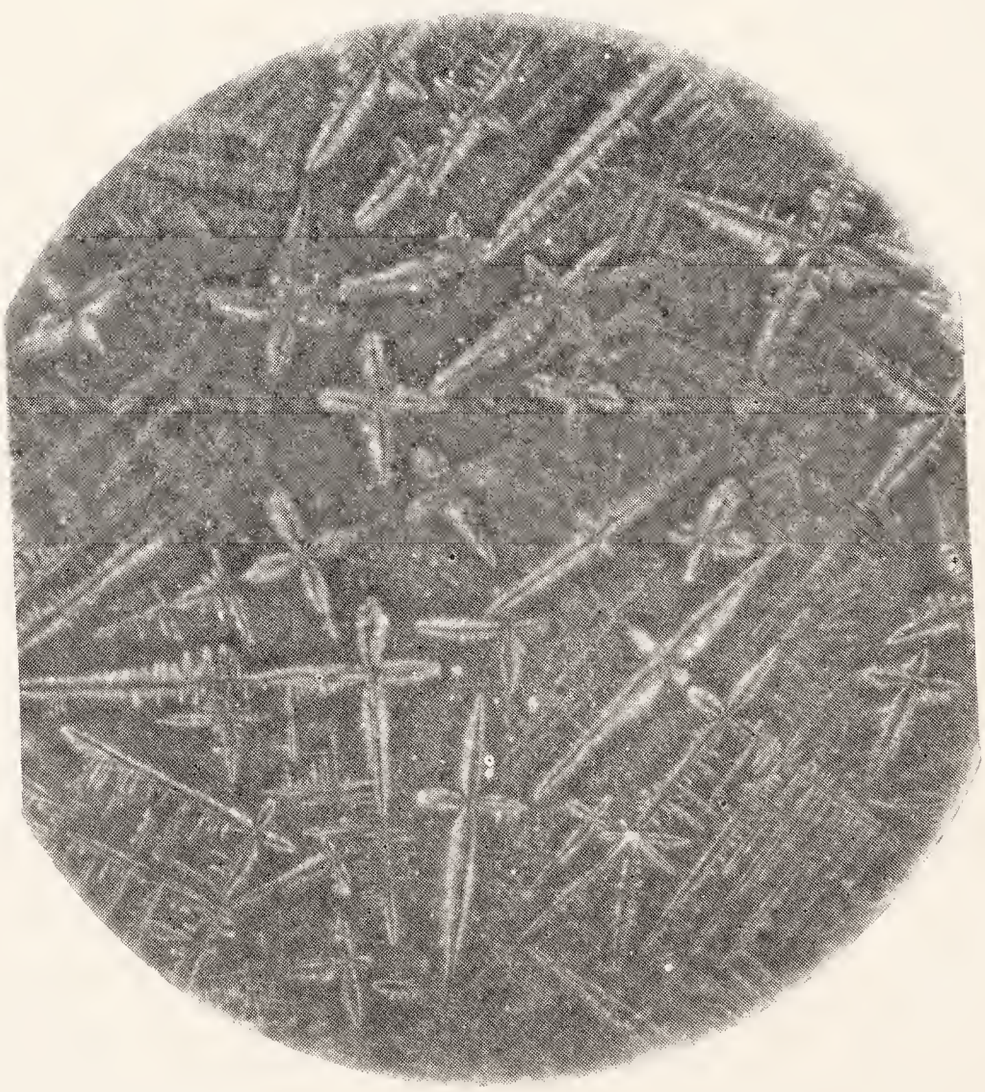

Fig. 6.

which had only appeared in a limited number in the former experiment (*Plate II, Fig.* 5). These experiments prove beyond question that urea is capable of combining with chloride of sodium in all proportions.

I had made a number of crystals which corresponded exactly with those I met with in the body, especially in the examination of sweat. I also made many intermediate forms which result from the combination of urea and chloride of sodium in varying proportion. I have not described these.

I next dissolved each of the dried microscopic specimens in a $\frac{1}{2}$ per cent solution of lactic acid. When they had dried again, I found that the crystals had undergone no change from their original form. I found further that they had become more stable and less deliquescent on exposure to air. I then understood how urea could exist in the presence of lactic acid, without being converted into ammonia, and how it could resist the acids it met with in the urine.

I next repeated my experiments by dissolving urea and chloride of sodium in distilled water rendered slightly acid by the presence of lactic acid. I kept the solution in a water-bath at 100° F. as before, but I did not produce a chemical combination of the urea and chloride of sodium; instead of this, the urea combined with the lactic acid.

In this way I discovered a very important fact. Urea can only combine with chloride of sodium in an alkaline or neutral solution. If sufficient lactic acid is present to render the solution even slightly acid, the urea combines with it to form lactate of urea, and becomes rapidly decomposed into lactate of ammonia.

Now, there are conditions of the body, especially during active exercise, when there must be a great

outpouring of lactic acid into the lymph, but yet the lymph preserves its alkalinity. We can now understand the way in which this is brought about. The lactic acid, by its combination with urea, causes the production of ammonia, which is the alkali necessary to preserve the balance.

If I have rightly interpreted my experiments, we should expect active exercise to be associated with increased excretion of ammonia. I shall, in another chapter, show that this result actually happens. We are thus in a position to understand better the methods, or at any rate one of them, by which the alkalinity of the blood is preserved. We can also understand another point: ammonia being formed will combine with any fresh lactic acid formed, and this combination causes the evolution of heat. We know that exercise raises the temperature of the body, and this must be, at least, one of the sources.

In the combinations made by urea with chloride of sodium, we have the true urate of the body, in contradistinction to the so-called 'urate of soda', which is an alleged combination of uric acid and soda.

There is no evidence of the existence of the latter salt in the body. I shall show that the crystals described as urate of soda by the earlier writers contained neither uric acid nor soda. The true urate is found in great abundance in the lymph, the sweat, and the urine; but in the latter it exists in such minute crystals that it appears difficult to prove that they are identical with the crystals I have produced by the combination of urea and chloride of sodium. The crystals in the urine are very minute, because urea exists there in great preponderance over chloride of sodium. In the normal sweat we find the dagger-shaped crystals with very

great frequency, because the chloride of sodium exists in great excess to the urea.

This fact suggested to me a test by which I could assure myself that the minute crystals which cover the whole field when a drop of urine is evaporated are identical with the artificial urates I had made by adding an excess of urea to chloride of sodium. If I altered the proportion in the urine so that chloride of sodium was in excess, I then ought to be able to produce the dagger-shaped crystals. I thereupon added chloride of sodium in excess to a specimen of urine and maintained it at 100° F. for thirty minutes. A gas was given off, showing that chemical combination was taking place. On evaporation I found abundance of the dagger crystals in the field. I also found that the minute urates had disappeared. They had become disassociated to provide the urea to combine with the larger amount of chloride of sodium. (*Plate II, Fig.* 6.)

This is a very complete demonstration of the chemical nature of the urate. The knowledge of its composition explains some of the gaps left in the story of nitrogenous metabolism. It explains how urea can exist in the lymph, side by side with lactic acid, without being converted into lactate of urea and passing into ammonia. The urea is protected by its combination, and is thus able to pass through the kidneys and exist in the urine without undergoing decomposition.

The fact that this combination cannot take place in the presence of free lactic acid, when the free urea combines with the lactic acid and becomes converted into ammonia, explains one of the chief methods used by nature to preserve the alkalinity of the blood, and also explains why the quantity of urea is diminished

under circumstances in which, without this knowledge, we should expect an increase. I shall refer to this question later.

We shall now look upon urea, not merely as the final result of nitrogenous metabolism and the possible cause of pathological conditions, as a product which we should be better without, but as an agent performing necessary functions and acting as a continual safeguard against the grave results which would follow if the lymph became an acid solution.

CHAPTER III.

LACTIC ACID.

Lactic acid has an intensely acid reaction, but it exists in the blood and the tissues without disturbing their alkalinity. The quantity present in any two individuals differs very widely, and there is also a great variation in the total amount present in any single individual at different times.

Although lactic acid circulates with the blood, the quantity which may be present in one part of a tissue at any moment may be out of all proportion to the quantity contained in another part contiguous to it. This acid therefore presents us with many points of physiological and clinical interest.

The physiological text-books do not give us much information respecting lactic acid. Sir Michael Foster (*Text-Book of Physiology*, p. 826) tells us :—

"The products of muscular metabolism pass into the lymph bathing the fibre, and so, either by a direct path into the capillaries or by a more circuitous course through the general lymphatic system, into the blood.

"The fate of the carbonic acid we have fully treated of in dealing with respiration ; the little we know concerning its nitrogenous product or products has been stated in dealing with urea ; the third recognized product is lactic acid or sarcolactic acid. Did any considerable amount of oxidation take place in the blood-stream while the blood is flowing along the larger channels, subject only to the influence of the vascular walls, we might fairly expect that the lactic acid discharged from the muscles would be subjected to oxidizing influences while still within the blood-stream of the larger channels. We have;

however, no satisfactory evidence of any lactic acid being oxidized in this way. On the contrary, there is a certain amount of experimental and other evidence that lactic acid present in the blood is somehow or other disposed of by the liver, and that if the liver fails to do its duty, lactic acid may appear in the urine."

If we turn to modern literature and consult the writings of those physicians who discuss the cause of an 'acid condition of the blood' and its effects upon the tissues, we find that they are so interested in uric acid, by which they explain all the symptoms, that the existence of lactic acid is ignored altogether.

This adds to our difficulties, because, while we cannot examine an animal tissue or fluid without coming in contact with lactic acid, if we want to investigate the subject of uric acid we must turn, not to the human body, but to the literature of gout and uric acid, for information. If we have any doubts about the fact that uric acid exists in the blood we are referred to an experiment made before the middle of the last century, which is regarded as convincing. If we are convinced, we are still not out of our difficulties, because we do not know what happens when these two acids meet one another in the tissues or in the blood. There can hardly be a 'tug-of-war', because lactates have the property of dissolving uric acid and therefore must get the best of it. The older physicians attached great importance to lactic acid as a cause of rheumatism and kindred diseases. With the advent of the uric-acid theory, lactic acid as a source of acidity of the blood, and as an irritant to the tissues, disappeared from literature. This fact is of more than historical importance.

We can obtain much valuable knowledge respecting lactic acid from a well-known physiological experiment.

The legs of a dead frog are stimulated by an interrupted faradic current, and the muscles vigorously contract. After a time the contraction ceases. It is then found that the muscle has become loaded with lactic acid. If we wash this away, and again apply the current, the contraction of the muscle is resumed. This proves to us that lactic acid is a product of muscular metabolism, and that it accumulates in the muscle during fatigue and arrests the function of the muscle.

We are not on such sure ground when we come to the explanation, viz., that perfect contraction of muscle evolves carbonic acid gas, and that when the contraction is imperfect lactic acid is produced.

We may have a series of the most vigorous contraction of muscle both in the frog and the human being, and still lactic acid is formed. If we admit that lactic acid is a product of imperfect combustion, we cannot leave out of account the fact that it is formed when the nerve supplying the muscle is exhausted. This point is of clinical importance.

We know that under all conditions the supply of lactic acid is maintained, and the amount is increased by exercise. We know also that any condition which limits the circulation in a tissue may lead to the accumulation of lactic acid in that tissue. This may be the result of cold or injury either to the tissues or the nerve supplying them. Thus, rheumatism is likely to occur in a joint which has been sprained many months before. The impairment of the circulation remains for a long time after such injuries.

That impairment of the circulation and a locally lowered temperature of the part precede the rheumatic attack in such cases is a point easily demonstrable.

We may say that the power of lactic acid to accumulate in any tissue is in inverse ratio to the activity of its circulation. This is a clinical fact to be remembered.

The great affinity which lactic acid has for the animal tissues may be demonstrated by placing a piece of meat or tendon, or—better still for purposes of demonstration—a small fish, into a 5 per cent solution of lactic acid. After it has been there for a few hours, pour off the lactic acid, and wash the meat or fish with cold water to remove the lactic acid from the surface. We can prove that the lactic acid has invaded every cell in the tissues, by the fact that the fish is permanently preserved against putrefaction. Thus we may keep it for months without any decomposition taking place, and this refers not only to every tissue of the fish but to the contents of the intestine. This also demonstrates the fact that one of the functions of lactic acid is to prevent putrefactive changes taking place in any dead organic matter which may be present in the tissues.

We have dead organic matter in the form of the dead cells, and that lactic acid plays a part in their removal we have seen in the previous chapter. From its great affinity to the tissues, and from some experiments I shall describe, we cannot imagine lactic acid existing in the lymph without invading the dead cell, and because it does so it preserves it from putrefaction.

Lactic acid is usually described as forming salts with zinc and calcium. It appears to have escaped attention that a large proportion of the salts we find in the excretions are either lactates or contain lactic acid. I have already mentioned that when present in excess it can combine with urea to form lactate of urea, and that this becomes transformed into lactate of ammonia.

It also combines with chloride of ammonium to

form lactochloride of ammonium. I have never seen chloride of ammonium in the excretions; it always appears as the lactochloride.

Lactic acid with chloride of sodium forms rods which appear either as crosses with rods of unequal length, or as a long rod with lateral rods at right angles and of unequal length. It closely resembles the lactochloride of ammonium crystal; both these are very common in the excretions of the skin (*Fig.* 7). Lactic acid forms with phosphate of lime a characteristic crystal which I shall describe later. Phosphate of lime

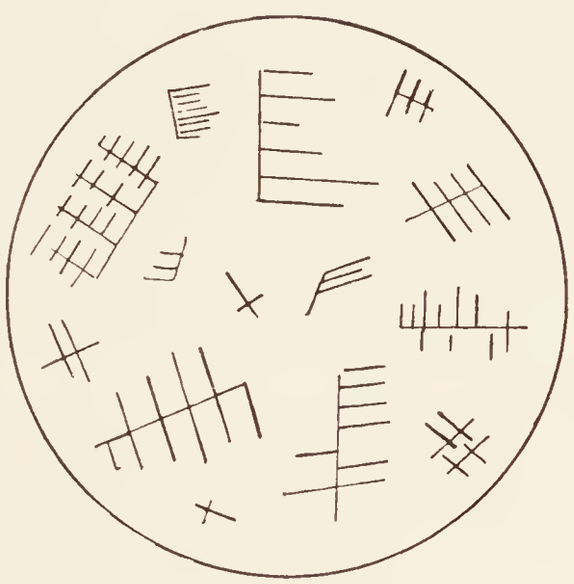

Fig. 7.

never occurs in the body except as a lactophosphate.

But the combinations of lactic acid have certain peculiarities which it is necessary to understand before we can interpret certain clinical and physiological results. The chemical combination alters the shape of the crystal, and after that has taken place more lactic acid may be absorbed into the crystal without any alteration taking place in its form. Thus lactochloride of ammonium may be an alkaline salt or an acid salt, according to the amount of lactic acid it

contains. The same is true of the lactophosphate of lime. Some part of this lactic acid exists in chemical combination, the remainder is additive; it remains in the crystal in a state of physical cohesion. We can illustrate this physical condition by prolonged trituration of one part of aniline violet with 1000 parts of powdered glass. We shall then have a pale lavender-coloured powder, which may be placed in water and kept there indefinitely without any solution of the aniline. This is because the energy of the cohesion between the particles of the aniline and the glass is greater than the energy of the water to overcome this cohesion. Now if we supply energy in the form of heat, and boil the water in which the aniline trituration is placed, the energy of cohesion is overcome and the aniline is dissolved and colours the solution. I think this exactly represents the physical condition of the surplus lactic acid in the salt. If we supply energy in the form of heat, we release the lactic acid from its cohesion and set it free.

If we take the fish which we have caused to contain an excess of lactic acid in a state of physical cohesion, and we place it in a solution of phenolphthalein which has been made red by the addition of soda, we shall find that this fish, charged with an intensely acid body, does not alter the alkalinity of the solution. It still remains red. Now if we apply heat to the solution, the lactic acid will be set free and the colour will be discharged from the solution in consequence.

This experiment also explains how it happens that an intensely acid body like lactic acid can exist in the lymph without altering its alkalinity.

Lactic acid is a very hygroscopic fluid, but its action on animal tissue is to make it lose its natural moisture

and to become rigid. It does this without effecting any alteration in the structures of the tissues it invades. This stiffness of the muscle is met with in practice under two conditions : one is after severe exercise, the other a short time after death. We call the latter condition 'rigor mortis'. Both are due to the action of lactic acid on the tissues.

'Rigor mortis' is ascribed to coagulation of the myosin of the muscle, but this explanation does not account for its subsequent sudden disappearance. We know that death releases a large amount of lactic acid which has been locked up in the tissues, and this free acid will at once invade the muscular and fibrous tissues as it did in my experiments. It thus produces the rigidity which is one of its characteristic effects. But it will also subsequently combine with the urea to form lactate of urea, and, as this decomposes, ammonia is formed, the lactic acid is again locked up as lactate of ammonia, and the rigidity disappears.

In the stiffness of the muscles which occurs after severe exertion, the lactic acid becomes gradually oxidized, and as it does so the stiffness disappears ; but owing to the increased acidity produced by the process of oxidation, the tissues become tender, and there is pain on movement. It may take twenty-four hours before these symptoms appear, and sometimes longer ; it may be some days before the process is completed and normal conditions are resumed.

As the excretions contain so many lactates, it follows that lactic acid must be a *normal* constituent of the urine. But lactic acid is never included in the chemical analysis of urine. It is described as sometimes existing in the urine under abnormal conditions. It is extraordinary that although the urine has formed the subject

of continuous chemical experiment for a century, and most of our knowledge of nitrogenous metabolism is derived from these experiments, the ingredient which is of distinctly the greatest clinical importance has escaped observation altogether; on the other hand, uric acid is regarded as a normal constituent, although it is a physical impossibility for uric acid to exist in the urine, except in minute quantities, because of its insolubility. It is obvious, without any chemical test being made, that the urine must, normally, contain lactic acid.

Uffelmann's test, which is a 1 per cent solution of carbolic acid, with sufficient ferric chloride to produce a purple colour, is the recognized test for lactic acid. If a drop of this solution be added to a drop of urine the colour immediately disappears. But this test is not altogether convincing when used in such a complex chemical compound as the urine. I have devised another test which is both interesting and instructive. Knowing the peculiar affinity which lactic acid has for animal tissue, I placed a piece of meat in some urine and allowed it to remain for some hours. I then removed the meat by the piece of string I had placed round it, and, after washing it under the tap, hung it up to dry. Although it was hot weather, and the meat was fully exposed, it showed no symptoms of putrefaction. It became dry and rigid as all animal tissues do which are immersed in a lactic acid solution. This is a conclusive proof that lactic acid exists in the urine and in large quantities. Incidentally this experiment shows that the affinity of lactic acid for animal tissue is stronger than its affinity for the salts with which it is in additive combination. The large amount abstracted shows that it could not have existed in

chemical combination. Some months later I used this piece of meat for another interesting experiment. I added sufficient soda to a solution of phenolphthalein to give it a brilliant red colour. I placed my meat in the solution and left it there for six hours. There was no change in the colour of the solution, showing that the lactic acid was locked up by its combination with the meat. This was a repetition of the experiment I have already described when meat was soaked in a 5 per cent solution of lactic acid, but it gives further proof that lactic acid had been abstracted from the urine. I then tested the further characteristic of lactic acid by heating the solution. When I had applied considerable heat, the colour of the solution disappeared, showing that lactic acid had been given off. I left the meat in the solution, and was surprised to see the next morning that the red colour had returned to it, the solution having resumed its alkalinity. The lactic acid had evidently gone back to the meat as the solution cooled. A very small amount of heat was necessary this time to set free the lactic acid again.

This is a very remarkable demonstration of the physical affinity which two substances may have for one another, and the way in which chemical changes can be produced by purely physical causes.

These experiments, in addition to proving that lactic acid exists in large quantities in normal urine, show how easily we can manipulate lactic acid and free it from its additive combinations, by heat.

There is another simple test for the presence of lactic acid in urine based on the same principle as the last. A solution of phenolphthalein is added to the urine, and then sufficient soda to render the urine alkaline, so

that a red colour is produced. If the urine is now heated, lactic acid is set free and its red colour disappears. I do not think that the fact that the acid reaction of the urine is increased by heat has been hitherto observed.

I have described the physical effect which lactic acid has upon animal tissues, how it causes them to become stiff and rigid, and this condition is permanent. On the other hand, we have seen that when lactic acid invades the living muscles, the stiffness it produces disappears in a little while, and is followed by more or less tenderness and pain in the part. Also that when lactic acid is set free in the body, after death, it causes a rigidity of the muscles which we call the 'rigor mortis', and this disappears as soon as the lactic acid has been locked up again by its combination with ammonia.

It might be thought that we might expect the same thing to occur in the dead tissue we have immersed in lactic acid, especially as lactate of ammonia is formed in that tissue. But we must remember that the quantity of urea existing in the tissue is limited, and, when all of it has been used to make lactate of ammonia, the quantity of lactic acid is not sufficiently diminished to materially alter its physical effect upon the tissues.

Now if we are influenced by the fundamental physiological error which leads us to suppose that every agent is only capable of producing one effect or series of effects upon the organism, and these effects can be increased by augmentation of the dose, we should expect that we could increase the rigidity of the tissue by using a stronger solution of lactic acid. If, instead of a 5 per cent solution, we used one of 25 per cent, and immersed a small fish in it, the results would

entirely differ. We should find the tissues soft and gelatinous, and instead of rigidity we should find the tissues would tend to break up as we handled them.

This may convey the idea that lactic acid had a direct destructive action on animal tissue such as the stronger inorganic acids possess. But if we look at the eye of the fish that has been so treated, we shall see that we are wrong in this conclusion. The cornea remains as translucent as it was during life. We know that every fibre and every cell has been invaded by lactic acid in large quantities, but not a single cell has been destroyed. We might think the effects were produced by coagulation, as is always suggested to account for the 'rigor mortis'; but we cannot adopt that view and account for the transparency of the cornea; neither does it help us to explain the peculiar transparency of the muscles and the tissues which takes place when a lactified piece of animal tissue has been exposed to the air for some time; on the contrary, it makes the theory of coagulation impossible.

I have not described the actual chemical combinations which lactic acid makes with the tissues, or the reason why it causes rigidity when weak solutions are used and great friability of the tissues when we employ stronger solutions, because this will best be considered in the next chapter.

CHAPTER IV.

THE GENESIS OF THE LITHATE.

The concretions found in and around the joints in patients of the lithic diathesis are practically insoluble, and occupy fixed positions from which the great physical pressure brought to bear upon them during the use of the joints fails to dislodge them. Now the urate, as we have seen, is a highly soluble body, possessing less tendency to concretion than most salts. It moves freely in the blood and the excretions of the body.

My imagination has never been equal to the task of finding any salt of urea which could produce an insoluble body such as we find in the joints. This is my reason for using the word 'lithate' instead of 'urate'.

The chemists have made two urates in the laboratory —an acid urate or biurate, which is prepared by boiling an alkaline carbonate with uric acid; and a quadriurate, which is hyperacid and is prepared by boiling uric acid with dilute solutions of acetate of potassium. Of these two salts the biurate is the less soluble, and is credited with being the urate deposited in the joint. But clinically we have to deal with a concretion which remains in the tissues for months or years, subject to all the solvent processes of the body, and to describe this as consisting of the less soluble of two very soluble salts has always appeared to me to throw a great strain upon our credulity.

If we introduced crystals of either of these urates into the tissues they would not remain there for five minutes.

Even if we accepted the view that the concretion was a urate, and through some mysterious process it had become insoluble, we should still have to account for its fixed position in the tissues. We should have to endow the crystal with the capacities of a limpet in order to account for it.

To understand the lithate we must go back to the dead cell. When I stated that urea was the ultimate product of the cell, I referred to its protoplasmic contents. I have so far made no reference to the wall of the cell, which is constituted differently from the cell-contents, and therefore its katabolism must take a different form.

Before we consider this point it is necessary that we shall have a clear idea of the nature of the cell-wall. Kölliker described it as follows: "The membrane of cells is mostly very delicate, smooth, scarcely capable of being isolated, and bounded by simple outlines, more rarely of considerable firmness and measurable thickness. Amongst the latter, we must include the cell-wall of the cartilages and fibrous tissue, which are thicker than those of other tissues".

But if we accept this idea of the cell-wall, and also that all tissues are built up of cells, how are we going to explain the great tenacity of all tissues? How do the cells that build up a tendon hold together when a great strain is put upon it during physical exertion? We must regard the cell-wall not merely as covering to the cell, 'resembling fibrous tissue', but as the cement which binds them together.

Just as a house would fall to pieces during the first wind which blew upon it except for the mortar which holds the bricks in place, so would the tissues fall to pieces unless their cell walls acted as a strong cement

to hold them together. Mortar is largely composed of lime, and nature uses the same substance to act as a cement in the case of the cell-wall.

The form of lime used is the phosphate, and the affinity of lactic acid for lime is so great that we cannot imagine phosphate of lime to exist in the presence of lactic acid without combination taking place. We never find phosphate of lime in the body except in combination with lactic acid. If we dissolve phosphate of lime in lactic acid and allow a drop to evaporate on a glass slide, we shall obtain a translucent substance which resembles varnish, and has the same adhesiveness which a varnish possesses when it is nearly dry. It is what the mechanic calls 'tacky', the condition in which he likes the glue to become before he joins two pieces of wood together.

When I described the great affinity which lactic acid had for animal tissue, it was really the attraction exerted by the lime in the cell-wall which was referred to. It combines with this lime in large quantities without in any way injuring or invading the contents or the cell. It is because of this that it causes no destruction of tissue.

The lime of the cell-wall serves another useful purpose in neutralizing the acidity of lactic acid. Like ammonia, it has quite remarkable powers as an antacid in relation to lactic acid. This helps us further to understand the way in which nature preserves the alkalinity of the tissues and the blood.

It does not follow that all the lactic acid in the cell-wall is in chemical combination with the lime. I have demonstrated by experiment that the cell-wall is able to hold very large quantities of lactic acid, and in such a state of cohesion that the acid is unable to

destroy the alkalinity of the fluids which surround it. We are sure that a large portion of this lactic acid is held by simple cohesion, because the action of heat alone is able to liberate it. The stiffness and rigidity of tissues produced by lactic acid appears to be due to a mechanical increase in the thickness of the cell-wall due to its presence. The cell-wall must swell owing to the increase in its contents, and there is no evidence of any chemical change taking place, as the excess of lactic acid can again be liberated by the simple action of heat. We can now understand how it is that urea can combine with sodium chloride in the presence of lactic acid. The lactic acid is locked up by the lime in the walls of the cell, so that the chemical reactions of the protoplasm within the cell are not interfered with.

We also can better realize why the dead epithelial cell is so difficult to dislodge in the absence of the lactic acid contained in the sweat, which acts as a solvent to the 'cement' which holds it there, as we have seen in our experiment with strong lactic acid solution. The tissues become friable and readily fall to pieces, because lactic acid has dissolved the cement which holds them together; but even under these conditions, lactic acid has not invaded the cell itself, or destroyed it, as was made evident by the continued transparency of the cornea of the lactified fish.

Lactophosphate of lime as we find it in the excretions is a perfectly soluble body. It appears under the microscope as round or oval dark bodies, of varying sizes, the smallest being larger than the urate. This is how it appears in relation to other bodies in the field. But if we change the focus so as to examine the structure of the individual body, we find that it is not a single

crystal, but a nest of very minute crystals in close cohesion. Here we have a very striking difference between the urates and the lime salts. The urate always appears as a distinct crystal, keeping a space between it and the next crystal. If we concentrate the solution so that this spacing is not possible, the urates will then appear each as distinct as pebbles on the sea-shore. Lime, on the other hand, never appears except in small concretions, no matter how dilute we may make the solution.

No amount of chemical research will reveal these physical facts, which are very important both clinically and physiologically.

A solution of lactophosphate of lime can be made by simply dissolving phosphate of lime in lactic acid. I have also prepared it by boiling the tendons of animals in lactic acid. On comparing the crystals of both these solutions with the crystals of lactophosphate of lime which one meets with in abundance in the sweat of rheumatic patients, I find them identical, not only in form, but also in the peculiar characteristics I shall now describe.

If we evaporate a drop of lactophosphate of lime on a glass slide, it has the appearance I have already mentioned (*Plate III, Fig.* 8).

If we leave it for twenty-four hours we shall find it has begun to sprout like a seed germinating, only instead of a single sprout there are a number, because each little crystal in the mass is, as it were, germinating. We thus get the peculiar bodies which are illustrated in *Plate III, Fig.* 9, and at a later stage the condition shown in *Plate III, Fig.* 10.

If we keep the preparation for some days, we shall find that each of these little sprouts develops into a rod-

PLATE III.

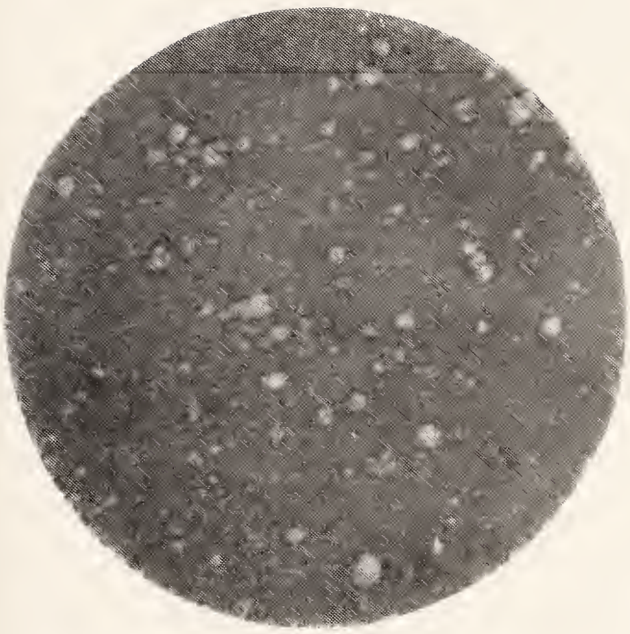

Fig. 8

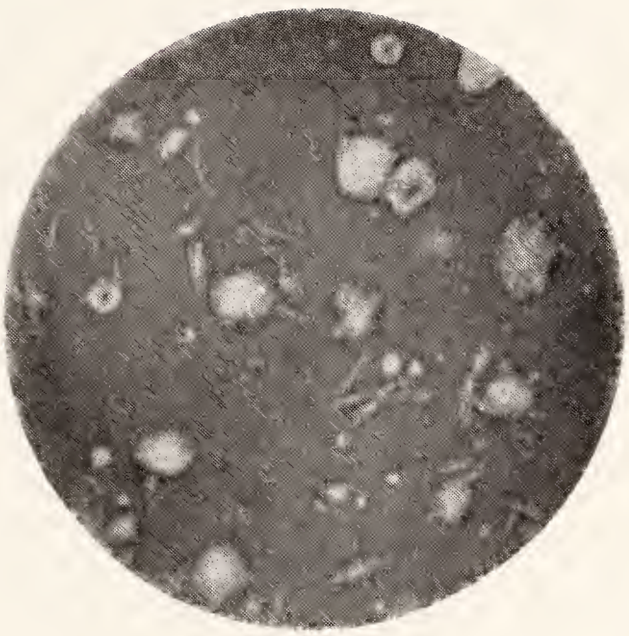

Fig. 9.

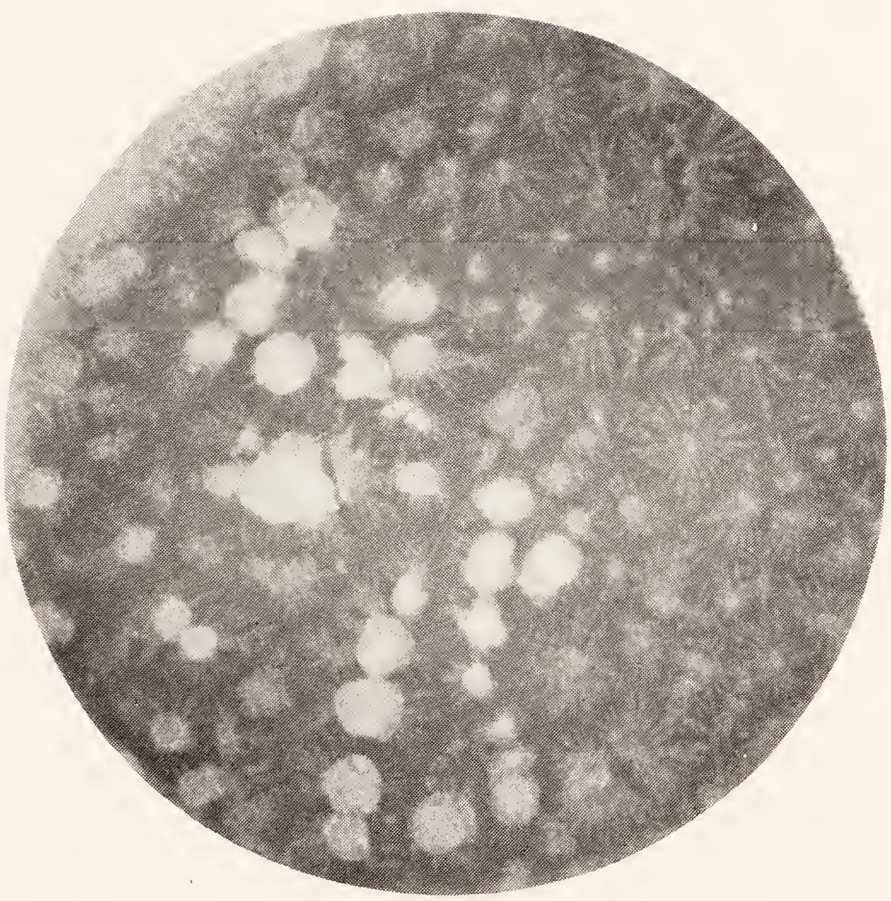

Fig. 10.

PLATE IV.

Fig 11.

Fig. 12.

like structure, and as these are pushed out from a common centre, the result is that we get a central nucleus with rays in all directions like the spokes of a wheel. Or we may have these rods only at two ends of the central nucleus, so that it looks like a bundle of sticks tied at the centre (*Plate IV, Fig* 11). Finally the rods break off from the central nucleus and appear as in *Plate IV, Fig.* 12, and become gradually shorter as oxidation progresses.*

Hydrate of lime goes through the same transformation, but instead of the rods being straight like sticks, they branch as they grow, and the branches can be carried to a great length from the original crystal. This will explain the remarkable binding properties of lime ; not only have its own particles a great tendency to cohesion, but by their branches they can bind together any other matter which may exist in the mass (*Plate V, Fig.* 13).

During this process the solubility of the phosphate of lime steadily diminishes, so that without other influence than the air to which the drop of lactophosphate of lime was exposed we can see a soluble body becoming almost insoluble. We see, therefore, that the normal constituents of the cell-wall provide us with all the material for the manufacture of the lithate. It is wholly unnecessary for us to imagine that the blood brings it there and deposits it.

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*These plates are made from negatives taken on paper. They are therefore complete as soon as developed. This method saves a great amount of time when a large number of microphotos have to be made. The difficulty is to secure a suitable paper. The Imperial Dry Plate Co., of Cricklewood, London, N.W.2, have made a large number of experiments on this point, and at last were able to supply me with a very rapid paper. I am much indebted to them for the great trouble taken to assist me.

But the reader will very naturally say that the deposits found in the joints of patients with gout have been proved to consist of 'urate of soda', and this has been confirmed by chemical analysis. I will leave the chemical part of the question until the next chapter; here we are most concerned with the physical properties and microscopic appearances of these deposits.

Garrod, in his work on *Gout and Rheumatic Gout*, furnishes us with some excellent illustrations of the crystals which he found in the joints, and to which he gave the name of urate of soda. *Plate V, Fig.* 14 shows crystals in the stellate form which it would be impossible to reproduce by any combination of urea, and which are characteristic of the formation of lactophosphate of lime in a certain stage of oxidation. In the same illustration we find the tiny rods which occur when a higher state of oxidation has been reached.

He also gives us another illustration which he describes as urate of soda, in which we see the stellate crystals of lactophosphate of lime after the rods have broken off (*Plate V, Fig.* 15).

Garrod was not alone in making this remarkable histological mistake. Thus, in Golding Bird's classical work on urinary deposits I find some excellent illustrations of lactophosphate of lime in its various stages of oxidation described as 'urate of soda'. *Fig.* 16 represents the nest of crystals as they are commencing to sprout; *Fig.* 17 shows the same in an advanced stage of oxidation.

It is only necessary to compare these illustrations with the photographs I have made of lactophosphate of lime in its various stages to recognize the mistake that has been made. There can be no question of a similarity between the crystals, because urate of soda

PLATE V.

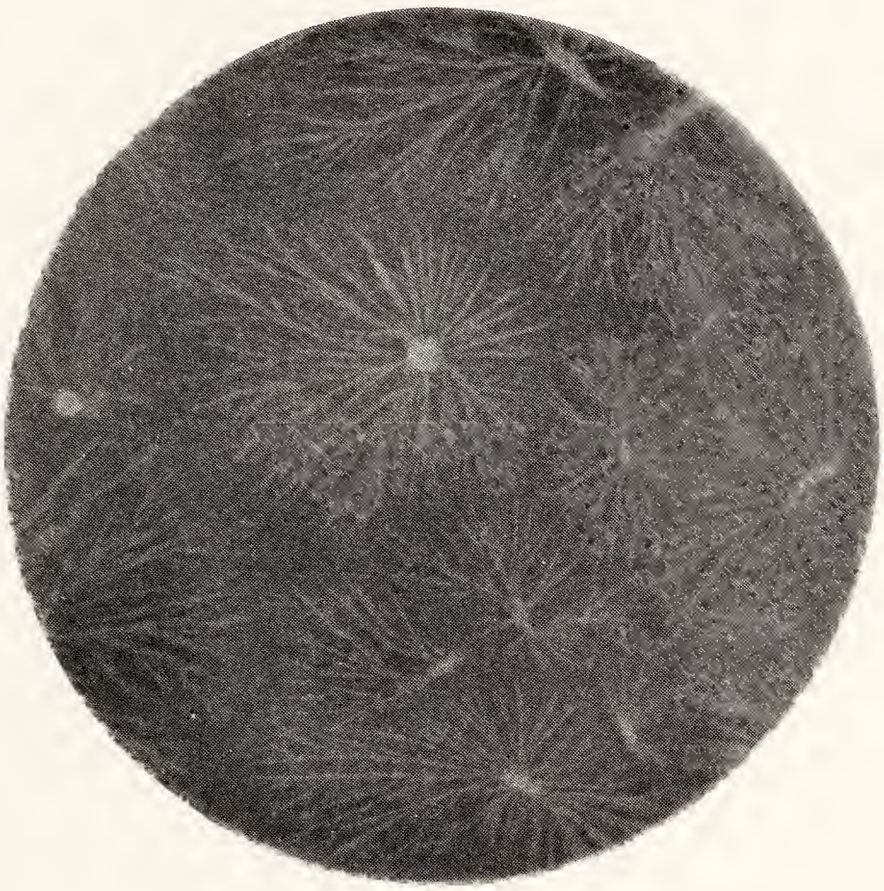


Fig. 13.

Fig. 14.

Fig. 15.

does not exist in the body, and cannot do so, for reasons I shall presently explain.

From what I have already described about the remarkable tenacity of the dead cell to the living cell, we have sufficient explanation of why these concretions remain constant in the tissues, in spite of all the forces brought to bear upon them. It remains for us to explain the physical causes which lead to these deposits being formed, instead of the lactophosphate of lime disappearing as a soluble salt as it does normally.

It will be remembered that these concretions only

Fig. 16.

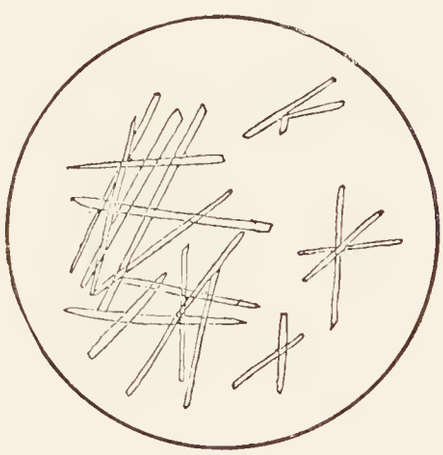


Fig. 17.

occur in the fibrous and cartilaginous tissues, never in the muscles or the other parts of the body. Now these tissues, which are the site of the deposits of lithates, occupy a position in the body which is practically outside the sphere of the normal circulation. If we make a section of a limb, we shall find in the centre the bone, which has a proper vascular supply; next we shall find the large mass of muscles, with their own circulation, which enables them to become the site of the great oxidizing processes of the body. At the circumference of the section we have the skin, with its rich capillary circulation, with ducts

where important oxidizing processes are carried out. Between the skin and the muscles is a space, and the tissues in this space have practically no circulation of their own, but are dependent upon the lymph exuded from its walls for their nutriment. It is within this great lymph-space that we find the tissues in which the lithate forms, and it is also the place where the metabolism of the products of the tissues takes place. The chemical changes occurring there are little influenced by the processes taking place in the far-away alimentary canal, less than would be supposed by the active circulation in the blood-vessels immediately outside its walls. The conditions which effect the chemical changes are almost entirely physical, and, because these physical agencies are necessary to perfect metabolism, the site selected for them is near the surface of the body and covers the whole area beneath that surface.

An internal organ, such as the liver, would lack all the essential conditions necessary to the processes which take place.

What happens in the lymph-space is easily intelligible. When the body is at rest, and the temperature normal, there is a slow exudation of lymph into the space, which nourishes the tissues within and supplies the necessary moisture for the performance of their functions. Very little lactic acid can pass into the space, because the muscles are inactive. The lymph, having performed its functions and taken up some of the products of katabolism, becomes itself a 'waste' product, and has to be eliminated. It finds its way by the lymphatics through a long and tortuous course to the thoracic duct, enters the venous circulation, and is finally eliminated by the kidney. If we now raise the temperature of the body while it is still in a state of rest, we dilate the capillary

walls of the space, and allow a much larger influx of lymph, which floods the space and brings greater nourishment to the tissues.

The body being at rest during the influx of lymph, there is no increase in the formation of lactic acid and its excretion into the space, but some of the additive lactic acid locked up in the lactophosphate of lime is set free. We have seen this happen in the experiments I have described.

The effect of heat on the body is to fill up the lymph-space with fluid and set lactic acid free; but this has an embarrassing effect upon the slow-moving lymphatics, which find themselves unequal to the work of eliminating the fluid as fast as it is poured into the spaces. This difficulty makes itself evident to the individual, who is conscious of the congested condition which results. But nature has made proper arrangements for the relief of this condition. The fluid and the lactic acid stimulate the functions of the sweat-ducts, which eliminate this excess of fluid, and immediate relief is obtained.

If we now consider the conditions produced by active exercise of the muscles, we shall find all the factors present which existed as a result of raising the temperature, but we shall in addition have a large accession of lactic acid owing to the activity of the muscle.

This free lactic acid, whether produced by heat or exercise, has a valuable effect in dissolving the cement which causes the débris of the dead cell to remain in contact with the living cell—because the cell is dead, lactic acid can produce its solvent influence. But the result of all this is that the lymph-space becomes crowded with fluid containing waste products, which must be quickly eliminated, or prejudicial effects will be

produced by the various chemical combinations which must take place.

Here, again, the skin by its excretion comes to our aid. The same causes which fill up the lymph-space with products requiring excretion, increase the excretory powers of the skin. The great result then of exercise is that the lymph-space has been flushed with fluid, more nourishment has been brought to the tissues, and waste products have been eliminated.

The whole scheme of nature is that the metabolism in the tissues shall have its periods of quiescence and its periods of activity, and that there should be those periodic flushings of the tissues in the lymph-space, just as we find them necessary for all purposes of cleanliness.

Under these conditions the lactophosphate of lime of the cell-wall passes away as a highly soluble salt, and is so unobtrusive that its existence has hardly been recognized.

But I commenced this book by recording observations upon a large number of healthy people where I found evidence of lithates in or around their joints, of which they were unconscious. These lithates indicated that they had collections of dead cells still attached to the living cells, and which had undergone partial oxidation so as to become nearly insoluble.

There was evidently something wrong with their physiological processes, although they were apparently in perfect health, took exercise, and made no errors in diet which I could discover. But I found that these individuals had two marked peculiarities: one was a dry skin that did not easily sweat either from heat or by exercise, and the other was a persistent subnormal temperature. Both these conditions may exist in persons in apparently robust health and who are

capable of great physical exertion. The conditions for producing lactic acid are the same in these persons as in others, but the power to eliminate it is deficient.

We have to consider what happens when lactic acid is thrown into the lymph-space and is not readily eliminated. We have seen from the experiments I have described that lactic acid has a very strong affinity for the cell-walls of the tissues, and if it is not eliminated will quickly combine with them instead of passing away with the lymph through the lymphatics.

Lactophosphate of lime as we find it in the lymph, the sweat, and the urine, is a very soluble salt, but it is soluble because there is sufficient lactic acid present to hold the lime in solution. Lactophosphate of lime as it exists in the normal cell-wall is not soluble, neither is it when we find it as lithate in the tissues. But the question of the decreased solubility of the salt is not wholly dependent upon its lactic-acid content. We have seen that the soluble salt becomes less soluble when exposed to the air, under the influence of oxygen. If we expose it to the action of sulphuric acid, which is at once an acid and an oxidizing agent, sparingly soluble crystals are at once formed. But, in spite of this fact, it attains its greatest insolubility under the influence of ammonia, which precipitates it from its solution. Therefore we are under no difficulties in explaining the presence of an insoluble lactophosphate of lime in the body, as we are if we try to represent the insoluble substance found as a urate.

That these lithates are characteristic of 'gout' can well be understood, because, as we shall see, there is not only a diminished production of lactic acid, but also there are conditions which cause the lactic acid to be locked up as lactate of ammonia.

But we should imagine that the presence of lactic acid would assist the detachment of the dead cell from the living cell. It does so under certain conditions. If the patient with a functionally inactive skin takes plenty of exercise and the body is kept warm, his lymph-space will be flushed with fluid containing lactic acid. This will dissolve up the débris of the cell-wall, and he will produce a quantity of lactophosphate of lime in a soluble form. But this has to be excreted, and, as the skin does not act, the whole work is thrown upon the lymphatics and, finally, the kidney. The result is that one of the most noted symptoms of 'gout' is that the patient passes large quantities of lithates in the urine, he may have gravel or stone in the kidney, and as an end-result the kidneys themselves become impaired. Some writers on gout have regarded this impairment of the kidneys as a cause of gout, instead of one of the final results of years of overwork and undue irritation.

When we find lithates in the joints of these patients, it will be usually in the great-toe-joint, or the shoulder, because neither of these joints gets that amount of exercise which is necessary to maintain it in normal physiological activity. Or we may find the urates in some joint which has at one time been injured, although the injury itself has been long ago forgotten, but the tissues have not recovered their full activity.

We shall find the lithates more abundant in the joints of those who, having a skin with defective functional power, take very little exercise, and so do not produce a sufficiency of lactic acid to dissolve the lactophosphate of lime. In these cases we shall not find the same tendency to pass lithates in the urine, because there is little to pass. The lactic acid which is formed combines with the lime in the tissues, and therefore we find in

such patients a large amount of lactic acid locked up in the tissues, so that its normal work is not performed. This fact can be demonstrated by the effect of the daily administration of baths which raise the temperature of the body. The lactic acid is slowly liberated, and the reaction of the skin shows a steady increase in its acidity.

We have to remember in these cases that the temperature of the patient is subnormal, and the active exercise, which might produce a temporary rise, is absent from the daily routine. Consequently, the lymph poured into the tissues is deficient, and there is not a sufficient surplus to act as a solvent, so the dead cell, slowly undergoing oxidation, remains in contact with the living cell, and presently we recognize its existence by finding crepitation in the joints.

Next to the failure of the action of the skin, cold and diminished temperature of the body are the most potent agents in the production of the lithates in the joints. Cold contracts the capillary blood-vessels and diminishes the flow of lymph, and the lymph-space becomes muddy from undissolved excretion.

CHAPTER V.

THE GENESIS OF URIC ACID.

Towards the end of the eighteenth century, Murray Forbes, observing the connection between gout and gravel, and the tendency of the gouty patient to form concretions, ascribed it to lithiasic or lithic acid in the blood. In 1848 Garrod described his well-known experiment. He added acetic acid to lymph, put it in a warm place for about forty-eight hours, and then found uric acid adhering to a piece of thread which he had placed in the solution.

This experiment proves that when lymph undergoes decomposition in an acid solution, it is capable of yielding uric acid. We know that when the constituents of lymph reach the kidney and undergo decomposition in an acid solution, they may produce uric acid, abnormally in man, but normally in birds. But Garrod asserted that he had found uric acid in the blood, and also that he had found it in the form of urate of soda. Garrod found, from a concentrated watery solution obtained from lymph and allowed to stand for some hours, numerous tufts of crystals, deposited on the sides of the vessel. He found that on the addition of hydrochloric acid they yielded rhombs of uric acid. When these crystals were incinerated they left an ash, alkaline in reaction, soluble in water, which did not answer to the tests for potash. He therefore pronounced them to be urate of soda (*Gout and Rheumatic Gout,* p. 98).

Neither Garrod nor any observer since has actually demonstrated the presence of urate of soda in the blood, but it has become universally accepted, and the uric-acid theory of gout is based upon it. From the descriptions in text-books it would appear as if urate of soda were simply the union of an acid with a base. Uric acid is really a very complex body, and has no affinity for soda.

If we place uric acid in a solution of carbonate of soda and maintain the solution at blood heat for a considerable time, no combination takes place; neither does the uric acid dissolve in the carbonate of soda, although we are always taught that carbonate of soda is a solvent of uric acid. It is not until the solution is *boiled* for some time that solution and combination take place.

Writers on gout tell us that uric acid is formed in the liver and immediately combines with soda to form urate of soda; some of this is carried to the kidney and some finds its way to the blood. Nature possesses no arrangement for boiling uric acid, and any delay in the combination would certainly result in uric-acid calculus in the liver, a disorder which fortunately never occurs.

The ordinary method of demonstrating the presence of uric acid in urine is practically the same as Garrod's test. A strong acid, usually hydrochloric, is added to urine. Now, as the urine is supposed to contain urate of soda, and uric acid is a weak acid, one might suppose that the strong acid would combine with the soda and the uric acid would be thrown out of solution; but this result does not happen. The addition of a strong acid to normal urine produces no visible effect. We must put it away in a warm place for twenty-four to forty-

eight hours, and then if we are lucky—for the experiment frequently fails—we shall find some uric acid at the bottom of the test-tube. The chemical change which actually takes place from the addition of the acid will best be explained when we have considered the following further method of demonstrating the presence of uric acid in urine.

The urine is saturated with chloride of ammonium, and then a few drops of liq. ammoniæ fort. are added. There is usually a very dense gelatinous precipitate, which is called urate of ammonium, and which has been given the formula $C_5N_4H_3NH_4O_3$.

This salt is of very great clinical importance, because it is used in the experiments by which the exact quantity of uric acid in the urine is determined.

From the results of these experiments we derive our knowledge respecting the influence of foods upon the excretion of uric acid. What is called the 'titration method' is used, and is based upon the oxidizing power of urate of ammonium on a standard solution of permanganate of potash.

"The urine is titrated with colloidal iron to remove an unknown substance that is precipitated by ammonium chloride, and made strongly alkaline with ammonia. The uric acid is rapidly and quantitatively precipitated as ammonium urate. This is filtered off, washed with ammonium sulphate to remove the greater part of the chlorides, dissolved in hot sulphuric acid, and titrated with standard potassium permanganate. The end point is reached when a momentary pink flush is seen over the whole body of the fluid." (S. W. Cole, *Practical Physiological Chemistry*, p. 341.)

In this experiment it is taken for granted that ammonia and chloride of ammonium have the property

of precipitating urates from solution. If we saturate a solution of urea with chloride of ammonium, and then add ammonia, we obtain no precipitate.

If we perform the same experiment with a solution of urate of sodium (i.e., uric acid dissolved in carbonate of soda), no precipitate falls. There is nothing to suggest that ammonia (NH_3) would have the property of forming an insoluble compound with urea or any salt of uric acid. Yet it is universally accepted that it does so. We know that chloride of ammonium and ammonia are the reagents used by chemists to precipitate the phosphate of lime and magnesia from solution, and the urine contains both these ingredients. If we use these reagents to an ordinary solution of sulphate of magnesia, we shall cause a white precipitate. Now it is absolutely impossible to add these ingredients to normal urine without causing a precipitate of lacto-phosphate of lime; it is also equally impossible to precipitate urea, or any salt of uric acid, with these reagents.

The answer to this objection is that the chemist can produce uric acid by adding an acid to this precipitate; but the inference that this is a proof that the precipitate therefore contained uric acid is open to serious question.

Chemically we have no proof that the precipitate contained any nitrogenous product whatever, beyond the ammonia which the chemist has himself added; and while this ammonia may be used for the synthesis of uric acid, it cannot precipitate uric acid already formed from solution.

To make myself clear upon this point, I added chloride of ammonia and liq. ammoniæ fort. to a specimen of urine, and produced the gelatinous precipitate called

'urate of ammonia'. I used the same ingredients in exactly the same way, to a solution of lactophosphate of lime, and produced a gelatinous precipitate. I

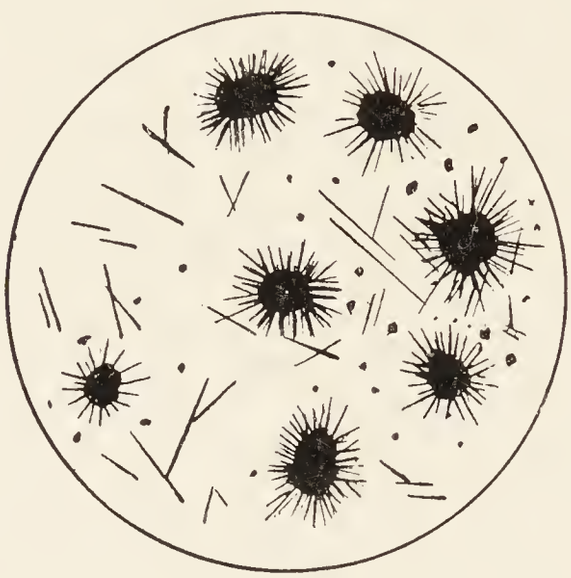


Fig. 18.

dissolved each precipitate in dilute sulphuric acid and examined them microscopically. The result is shown in the accompanying illustrations. *Fig.* 18 represents the

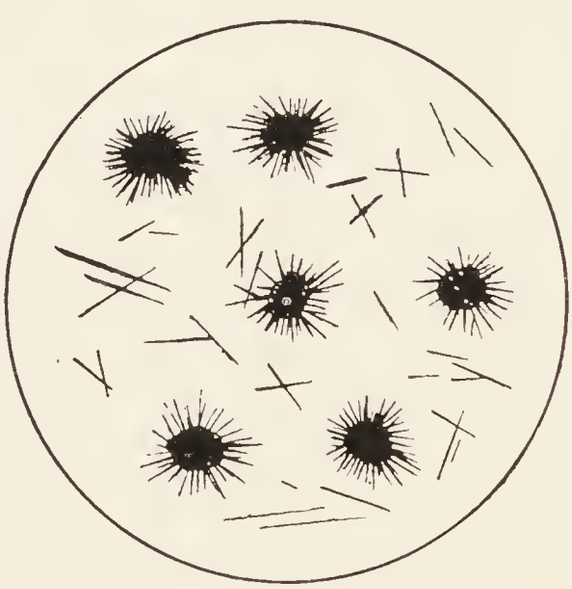


Fig. 19.

precipitate obtained from urine, and *Fig.* 19 that obtained from lactophosphate of lime.

In the former illustration there are some granular

particles which do not appear in the second. The precipitate obtained from urine by these reagents contains some of the colouring matter of the urine; and as this enters into the composition of the uric-acid crystal, and appears to be an essential part of it, the fact is of importance. The crystals I have illustrated above, due to the action of sulphuric acid on lactophosphate of lime, were noticed and illustrated by Golding Bird in his work on urinary deposits, and were described by him as 'urate of soda' (*Fig.* 20).

To revert to the 'titration method', the final result,

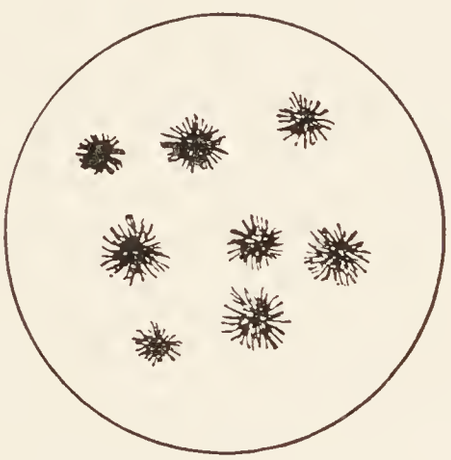


Fig. 20.

by which the amount of uric acid is determined, is obtained by dropping a standard solution of permanganate of potash into the solution, and there comes a moment when the colour does not disappear and a pink flush is seen in the fluid. But if we perform the same experiment with a solution of lactophosphate of lime, we shall obtain the same results. If uric acid actually existed in the solution, we should not expect the reaction, because it is a stable body and has not the chemical activity or oxidizing power of phosphate of lime.

It will be noticed that, throughout this inquiry, when we think we have come upon uric acid, or urates, we

invariably find ourselves in contact with lactophosphate of lime. For convenience I have prepared a diagram showing the changes which lactophosphate of lime undergoes when simply exposed to the air. As I have already shown, each of these varied forms has been described by one observer or another as ' urate of soda '. They have done so because they found that by adding an acid they could produce the rhomboid crystals known as ' uric acid '. (*See Fig. 27.*)

I have alluded to Garrod's experiment. He discovered tufts of crystals in a concentrated watery solution obtained from lymph, and he found that when they were incinerated " they left an ash, alkaline in reaction, which did not answer to the tests for potash", and therefore he concluded that it was soda. It did not occur to him that it was lime, and that what he called urate of soda was really lactophosphate of lime.

But now we come to a difficult problem. How does it happen that lactophosphate of lime in the presence of a strong acid, and after exposure to the air, yields ' uric acid ' ?

We have to remember that in all these experiments we are not dealing with a pure solution of lactophosphate of lime, but with a solution originally containing urea, which becomes converted into ammonia during the experiment, or with ammonia directly added by the chemist.

Thus, when Garrod " discovered uric acid in the blood ", he made the lymph strongly acid, and then left the lymph to decompose in a warm place for thirty-six hours or longer. The result was that the urea became converted into ammonia, and this in its free state has the property of precipitating lactophosphate of lime.

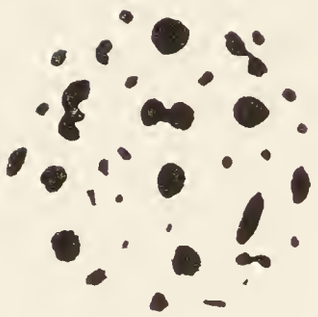


Fig. 21.

Fig. 22.

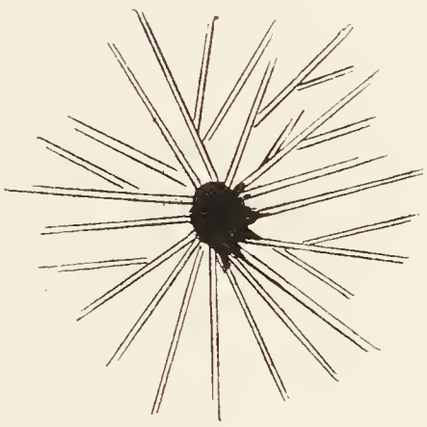
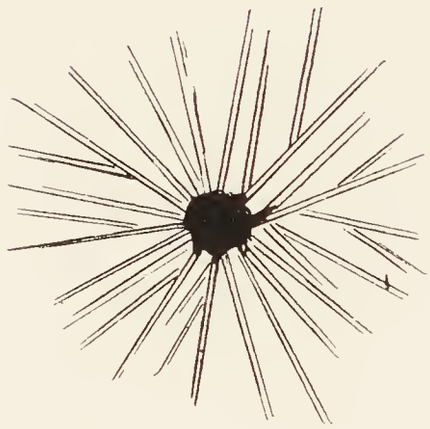


Fig. 23.



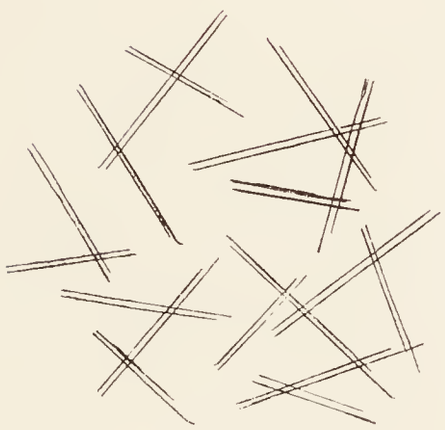


Fig. 24.

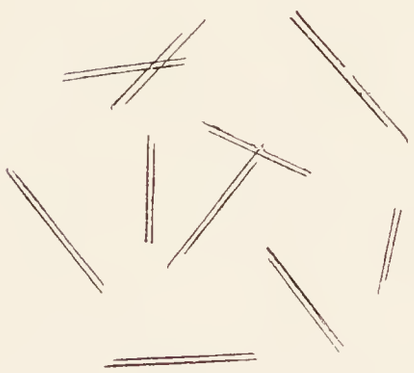


Fig. 25.

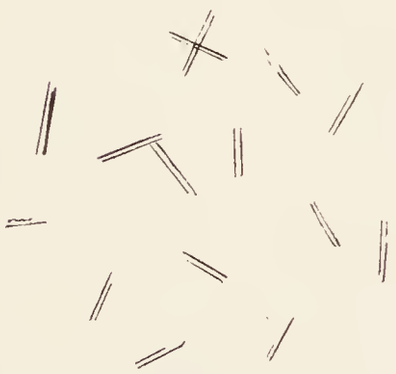


Fig. 26.

Fig. 27.

In the titration test we do not wait to manufacture ammonia from urea, but saturate the urine with chloride of ammonium, and then add some ammonia, and having precipitated the lactophosphates we redissolve them with hydrochloric acid. This all points to uric acid being formed by some reaction which takes place between lactophosphate of lime and ammonia in the presence of an acid and oxygen. It would appear from this that the synthesis of uric acid would be a very simple matter.

Not being a chemist, I have found it a very difficult problem, one of the difficulties being that the so-called urate of ammonium contains some of the colouring matter of the urine, and is not a simple compound of ammonia and lactophosphate of lime.

But the genesis of uric acid can be rendered intelligible from the following experiments.

We have seen that when lactophosphate of lime is exposed to the air for a prolonged period, it finally forms very small fine rods (*see Fig.* 26). If we add an excess of lactic acid to the solution, and then place a drop on a glass slide and add to it two drops of peroxide of hydrogen (20 vols.), the excess of lactic acid prevents evaporation. In about six hours we shall notice crystals being thrown out of solution, and these crystals are identical in form with those of uric acid—not only do we find the ordinary rhombs, but in varied experiments we obtain many of the less common forms of uric acid (*see Fig.* 27). Now it is a little difficult to understand the composition of these crystals. Uric acid does not contain either phosphoric acid or lime, but it does contain nitrogen. In this solution we have no nitrogen unless it has absorbed ammonia from the air; yet these crystals

are identical in form with uric acid, although they do not possess its insolubility.

The chemical problem can best be understood if we consider the solution in which chloride of ammonium is combined with lactophosphate of lime and then exposed to the influence of oxygen and an acid. When lactic acid is oxidized it is converted into carbonic acid gas, and this has a great affinity for lime. One of the first stages, therefore, would be the formation of carbonate of lime; but, as the solution is highly acid, this would be at once converted into the acid carbonate of lime, which has no tendency to crystallize. But the acid carbonate possesses the property of liberating ammonia from the chloride of ammonia, and we shall obtain both free ammonia and carbonate of ammonia in the solution. Now as a result of these chemical changes phosphoric acid must be set free, and as it is a very powerful oxidizing agent, containing as it does four atoms of oxygen, its effect upon the lactic acid would be to convert some of it into carbonic acid gas; but the conversion is not complete, because some lactic acid remains in the solution and forms a lactocarbonate of ammonium. It will be thus seen that although lactophosphate of lime is essential to the chemical changes brought about, it forms no part of the final product.

That these results are not purely theoretical can be demonstrated by the following experiments.

If lactic acid be added to a turbid solution of lime-water, the lime in suspension is at once dissolved and a clear solution is produced.

The same occurs when phosphoric acid is added to a turbid solution of lime-water. In both cases the lime is converted into a soluble salt.

Now if we warm lactic acid and phosphoric acid together, there is no violent evolution of carbonic acid gas, but if we add a drop of the mixture to a clear solution of lime, we at once produce a precipitate, showing that carbonic acid gas has been evolved by the combination. Thus we know that if by any means phosphoric acid is set free from the phosphate of lime in the presence of lactic acid, carbonic acid is produced. It will be seen, therefore, that the purpose served by the lime in the laboratory of nature is to set free ammonia from its combination, and that of the phosphoric acid to oxidize the lactic acid and convert into carbonic acid.

As a further test for my conclusions, I tried to produce the same result without the use of the phosphate of lime. My idea was that we should be able to do without the lime if we supplied an acid carbonate ready made, and without the phosphoric acid if we supplied the necessary oxygen. I made some lactate of ammonium by adding ammonia to lactic acid, and when chemical reaction had ceased I introduced some carbonate of ammonium, which caused great effervescence. When this had subsided, I added peroxide of hydrogen and applied heat. There was active chemical change, and when it had ceased I made the solution acid with hydrochloric acid. After leaving it for a time, I put a drop on a glass slide and allowed it to evaporate. When dry the whole field was covered with crystals of lactate of ammonium. I redissolved these crystals in peroxide of hydrogen, and in a short time I found crystals identical in form with uric acid thrown out of solution.

I allowed the preparation to dry, and after twenty-four hours a number of large crystals similar in shape

to uric acid, with decided markings, appeared. From previous experiments I recognized these crystals as being similar in composition to uric acid, but containing excess of ammonia, and quite soluble. I may remark here that while ammonia in excess increases the solubility of the uric-acid crystals, it renders the crystal of lactophosphate of lime less soluble; but in both cases the presence of ammonia does not of necessity alter the crystallization, but shows itself by markings

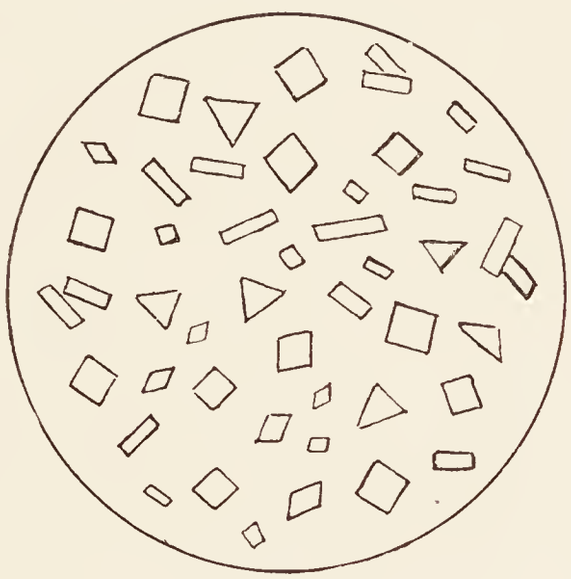


Fig. 28.

on the face of the crystals as if they had been roughly sculptured. These are not visible in the minute uric-acid crystals usually met with, but it becomes apparent when larger crystals are manufactured.

To the dry preparation containing these large soluble rhomboid crystals, I added strong hydrochloric acid. The immediate effect was the deposition of small uric-acid crystals in large quantities, insoluble in strong hydrochloric acid (*Fig.* 28). Now it is evident that in this experiment I had too much ammonia and not

enough acid and oxygen in my original solution, but this fact makes the results more instructive, as it shows more clearly the factors essential to the genesis of uric acid.

I do not wish it to be inferred from these experiments that uric acid is simply a lactocarbonate of ammonium, but I think we may be safe in saying that uric acid is formed from lactic acid, carbonic acid, and ammonia by the action of oxygen in an acid solution, and that before we can make a true synthetic product we require the addition of the colouring matter of the urine. If I were a chemist I might express myself better and give chemical formulæ.

I think the true importance of these experiments is to bring home the fact that while attention has been drawn to the insoluble form of uric acid, on account of its insolubility, there are salts containing practically the same ingredients which are soluble, and that in the human subject these salts are formed more frequently than the insoluble kind. I think it also shows that a highly acid solution is necessary to the formation of the insoluble form.

I think we may infer from these experiments that when dealing with a tendency to form phosphate of lime calculi, a vegetable dietary which may increase the excretion of ammonia is contra-indicated; and that in the tendency to form uric-acid calculi, the reverse is true. In fact, I have used chloride of ammonium with great advantage in cases where there was a tendency to excrete uric acid. This shows how very important it is not to regard chloride of ammonium as the agent which has the peculiar property of precipitating urates, as is stated in a recent work on physiological chemistry. I do not

wish the reader to infer that I regard chloride of ammonium as a "solvent of uric acid". There is no drug or chemical which can dissolve uric acid in the body. It is contrary to the whole scheme of nature, for reasons which I will explain. The effect of the chloride of ammonium is to prevent the formation of the insoluble salt of uric acid, which is quite another matter.

Chemists give uric acid the formula, $C_5N_4H_4O_3$. In Schäfer's *Physiology* (p. 586) it is stated that, "rightly to appreciate the physiology no less than the chemistry of uric acid, its close relationship to urea should be clearly understood. It yields the latter by a combined process of oxidation and hydrolysis". If this is true, the urea must be formed from the ammonium carbonate during the decomposition which takes place when uric acid is formed. Thus it is only necessary to take the elements of water from ammonium carbonate to form urea. But I have shown that there is no urea in the so-called ammonium urate from which uric acid is produced. If we take a solution of prepared urate of soda and add to it a few drops of Nessler's solution, we shall at once obtain an orange-coloured precipitate indicating the presence of ammonia. Therefore I feel justified in saying that uric acid contains no urea.

That uric acid contains lactic acid can be easily ascertained by boiling a little uric acid with some distilled water, and then adding Uffelmann's solution. The blue colour is instantly discharged.

We have now to consider the conditions which govern the formation of uric acid as a product of the human body. All the ingredients out of which uric acid is made exist in the blood; but the essential conditions

under which they can undergo the necessary combination to form uric acid are not existent, because the fluids of the body are alkaline and uric acid can only be produced in a highly acid solution.

But it is also part of the scheme of nature that the fluids of the body and the products of katabolism shall become acid immediately before excretion. For this purpose the kidneys and the skin are provided with the necessary apparatus for their oxidation.

It appears probable that this is a necessary precaution against the action of bacteria, which might easily find their way through the urinary organs to the kidney unless means were taken to maintain the excretion in a state of acidity.

Another reason is that while the excretory organs render the fluids which pass through them more acid by oxidation, this acidity stimulates the excretory power of the organ. This is an important clinical fact. It can be illustrated by the activity of the skin excretion in a case of rheumatic fever.

We know that in the human being the urea passes through the kidney unchanged, in spite of the process of oxidization and the acid medium which surrounds it. I have demonstrated that it is able to do this because of its combination with sodium chloride; but, just as we could break up this combination in the laboratory by the use of strong acids, so can an abnormal condition of the urine in the kidney disassociate the sodium chloride, set free the urea, and produce those changes by which ammonia is formed, which I have already described.

It does not follow that these chemical combinations will of necessity produce uric acid. Normally, in the human body, it does not do so. We may have a salt

formed which is soluble in the body and which will remain soluble when the urine is cold. Or it may be soluble at the temperature of the body and be thrown out of solution as the urine cools, and be again soluble when heat is applied. Or we may have a salt formed which is soluble in the body, but which is precipitated as the urine cools, and then being oxidized by the air becomes insoluble when heat is applied. Or we may have—and this more rarely in man—a salt formed which is insoluble at the temperature of the body and is excreted as a solid. This is normal in birds.

It is the less soluble combinations which have attracted attention. The more soluble of those which we call lithates or urates, are regarded as of little clinical importance, while the most soluble and most normal in man has attracted no attention at all. But this highly soluble body can be converted, as I have shown, into the insoluble salt by the addition of a mineral acid and by exposure to the oxidizing effects of the air.

Here we have the fundamental error of the whole uric-acid theory. Because the chemist can manufacture uric acid from normal urine, he has supposed that it already existed there. This conception rendered necessary the existence of some supposititious base with which the uric acid could be in combination and so obtain solubility. In this way the urates, biurates, and quadriurates came to exist in the text-books, but not in the human body. In the course of these investigations it will be noticed that clinical facts difficult to understand become perfectly intelligible and natural directly we reach the physiological truth. We can understand now why, if a man of sedentary habits takes hard physical exercise on a particular day,

on the following morning his urine is loaded with lithates. Some part of this may be due to the great lymph-space having been flushed with fluid, causing an increase in the solid matter excreted. But this does not explain all the facts. This matter might be passed in a fluid form, and we should only notice an increase in the specific gravity of the urine. But we may have a subnormal specific gravity in a urine which deposits urates directly it cools, and we may also find crystals of uric acid in the deposit. This is easily intelligible when we remember that vigorous exercise causes higher oxidation, and that the ingredients which enter into the composition of uric acid and urates are consequently rendered less soluble. This also explains how it is that foods, which contribute nothing to the ingredients of which uric acid is composed, have been proved guilty of increasing the quantity of uric acid. Red meat, sugar, and alcohol increase the oxidizing power of the body, and therefore result in an augmentation of the salts excreted from the body in an insoluble, or partially insoluble, form.

We know that birds, who do not indulge in alcohol, sugar, or an excess of red meat, have a urinary excretion which consists of little else but uric acid. But, owing to the large amount of exercise they take in the fresh air, their katabolic products must be very highly oxidized, and they must also form a large amount of lactic acid. The urine of birds is passed in an almost solid form, and it is difficult to understand how this condition is compatible with its passage through the kidney. I think I have found the explanation in the following experiment. Uric acid is very soluble when boiled with liquor potassæ. If a strong solution of uric acid is prepared in this way and some peroxide of

hydrogen is added, the solution is converted into a semi-solid mass. This occurs instantaneously, and it is easy to suppose that this chemical change takes place at the moment of excretion. The sudden liberation of phosphoric acid would provide the necessary oxidizing agent.

We may now ask what purpose uric acid serves in the economy of nature. I know that we are taught to regard the products of metabolism not only as waste matter but excrements wholly pernicious, and that uric acid is regarded as the worst offender. But we have seen that urea has useful functions to perform in maintaining the alkalinity of the blood. I have yet to discover any evil thing it accomplishes. Lactic acid, while capable of causing so much pain, performs functions which we have seen to be vitally necessary. Both these are soluble bodies capable of active chemical combinations. Uric acid, on the other hand, is insoluble, and cannot enter into combination with the salts in the urine or with the tissues.

This is the rationale of its existence. This will be better understood if we consider for a moment the method by which the chemist manufactures uric acid from the blood or the urine. He simply makes it strongly acid and leaves it exposed to the air, and then all the chemical processes by which uric acid is made go on automatically: slower under these conditions than they would do in the body, but still they take place.

Now suppose, when the urine has been rendered sufficiently acid to produce uric acid, instead of putting it aside in a warm place we inject it back into the bladder, the result would be a very violent inflammation produced by an excess of acid, and this would probably

prove fatal. But such a state of intense acidity might easily occur in the kidneys, because it is part of their normal function to increase the acidity of the urine, and there are many conditions which cause the urine to be extremely acid when it reaches them. Nature has to provide some means for checking this process if it goes too far. The method used in the lymph of making the increase of acid produce ammonia would not be satisfactory, because it is necessary that the urine should retain a large degree of acidity. The method adopted is, when a certain stage of acidity has been reached, to lock up the acids in an insoluble body which can be excreted without exciting any inflammatory effects upon the mucous membrane of the urinary organs.

It is for this reason that we find uric acid in urine which may not have a high degree of acidity because the acids are locked up. In such urines, if the acids were liberated from the uric acid, we should have a highly acid solution.

As we understand the nature of uric acid, we see the difficulties created by the false conception that uric acid exists in the blood ; when we regard it as a safeguard against hyperacidity of the urine, we can comprehend the purpose of its existence. From a clinical standpoint it is not the existence of uric acid in the urine, but its absence, which is of most importance. When uric acid appears in the urine we know that the lactophosphates are being excreted, and these are necessary to the manufacture of uric acid. It is the failure of this excretion which causes many serious symptoms. This makes the chloride and hydroxide of ammonium test one of the most important in the examination of urine. If we obtain a precipitate with

these reagents we need not trouble ourselves further, but if we obtain none or very little we know that the kidney is failing to excrete them. This failure may cause symptoms which are obscure until the cause is known. Thus, attacks of giddiness and vertigo, which occur both during rest and during exercise, are an indication to examine the urine, and, if we find diminished excretion of phosphates, we may be fairly sure it is the cause of the trouble. The same applies to some forms of headache and drowsiness, and also high blood-pressure. If the patient has lithates in the joints, the effect of the check of excretion will be to set up chemical changes in these lithates and cause symptoms of 'gout'. It is not uric acid which produces these symptoms, but the soluble lactophosphate of lime. The confusion between these two bodies, which differ so widely in their physical and chemical relations, creates a difficulty in understanding the clinical symptoms and the effects of treatment. The true position is this: uric acid begins its existence in the kidney, and then only under uncommon conditions, and terminates its connection with the body when the urine is excreted.

Lactophosphate of lime exists in every fluid, tissue, and organ of the body. It is broken up in the process of making uric acid, but this can only be accomplished in a very acid solution. It is the excretion of lactophosphate of lime which is of importance, and it is a curious fact that all the fluids used to estimate the excretion of uric acid contain lactophosphate of lime and no uric acid.

It will be observed that I have reached conclusions respecting the physiology and chemistry of uric acid

which wholly differ from the statements made in the extensive literature of the subject.

It would occupy too much space to enter into a discussion of the various theories put forward. They commence with the assumption that uric acid can be formed in the alkaline fluids of the body, and then go on to describe uric acid as performing physical and chemical feats of which an insoluble body is incapable. It is only on occasions that the supposititious soluble salt of uric acid is introduced. Thus, it is not until we come to the fourth chapter of Haig's work on uric acid that we are informed that "uric acid in the title of this book, and generally throughout the book itself, unless otherwise specified, is used as an inclusive term comprehending uric acid and its salts".

This makes the preceding chapters as intelligible as if a writer on therapeutics insisted upon calling Epsom salts sulphuric acid, because by certain chemical experiments he could obtain sulphuric acid from these salts.

But even with this explanation we are still left in difficulties, because Haig insists that the urate exists in the blood in an insoluble form. He says :—

"Sir Wm. Roberts says (*Urinary and Renal Diseases*, p. 73), 'It may be regarded as probable that the defective power of the kidneys to eliminate uric acid in gout arises from a diminished alkalescence of the blood'.

"From my point of view it has nothing whatever to do with the kidneys; the urates are not in solution in the blood, and are not brought to them; when they have been got into solution by an alkali they are excreted fast enough."

The urate, therefore, is considered by Haig as an

insoluble body which, as he frequently reiterates, can be rendered soluble by a single dose of alkali, and rendered insoluble (whatever this may mean) by a single dose of acid.

But Haig admits that his powers to produce this marvel have limits. On page 15 he tells us that "though I could produce a large excretion of uric acid at pleasure (by the administration of an alkali), I could not keep up the excretion very long. I could produce nothing that at all resembled an extra formation of uric acid".

As Haig's conclusions were based on numerous long and laborious experiments, it is of some importance to know the methods by which he reached them. It is only in the last pages of the book that these are described. He estimated the quantity of uric acid by Haycraft's method, of which he gives a complete description. To 25 c.c. of urine, 15 gr. of bicarbonate of soda are added (Haig used 30 gr.). It will be noticed that this differs from the titration test in the fact that in the latter the urine is saturated with chloride of ammonium to ensure its alkalinity; in Haycraft's test the amount of alkali added had no reference to the degree of acidity of the urine. I think this point has an important bearing upon the results obtained by Haig.

The sample is next treated with 2 to 3 c.c. of strong ammonia and about 2 c.c. of ammoniated silver nitrate solution. This brings down a gelatinous precipitate which has received the name of urate of silver. Now nitrate of silver has no more power to precipitate urea or urates than ammonia possesses, but both have the property of precipitating lactophosphate of lime. Therefore the urate of silver must be classed with the urate of ammonium as a chemical mistake. The

solution used by Haig contained no urea, nor uric acid, and the only nitrogenous product was the ammonia he added.

The precipitate, after being filtered and washed by a tedious process, is dissolved in nitric acid and water, has a few drops of ferric alum solution added to act as an indicator, and the ammonium thiocyanate solution is added until a permanent pink colour is produced.

The amount of 'uric acid' is estimated by the amount of thiocyanate solution necessary to produce this result. In both chemical methods the same mistake has been made, and as a result nitrogen, a vital necessity of nutrition, has been made to appear as a source of evil, and has been charged with crimes of which it is wholly guiltless.

CHAPTER VI.

EXCRETION.

The great lymph-space which lies between the skin and the muscles has been referred to, and the great importance of the skin as an organ of excretion to eliminate the waste products which are formed in or discharged into this space will be appreciated.

But in the study of metabolism, physiologists have proceeded on the assumption that all the waste products of proteid metabolism pass through the kidney, and that as a result we can estimate the amount of nitrogenous excretion by analysis of the urine. It is well known that urea can, and does, pass through the skin, but the quantity has been regarded as negligible. This basic hypothesis is distinctly fallacious, and its fallacy is never more apparent than when the skin is performing its functions actively, such as during vigorous exercise.

The wide difference between the results obtained and the actual facts is well known, but instead of this being regarded as a convincing proof that the method of experiment was erroneous, it has led to the introduction of new theories to explain the facts, with the result that an altogether unnecessary confusion has been imported into the whole discussion.

To make this difficulty understood, I will quote a few paragraphs from Schäfer's *Text-Book of Physiology*, p. 912 :—

"The most interesting question in connection with the special metabolism of the muscles which remains to be

considered, is the effect which their exercise produces upon the proteid metabolism of the body. It was the opinion of Liebig that the energy of muscular contraction was produced by the oxidation of muscular substance, and it would follow from this that the exercise of the muscles must tend, *ceteris paribus*, to increase the amount of nitrogen excreted in the urine. This doctrine of Liebig's was accepted for many years by physiologists, but was, for a time at least, completely overthrown by the results of the famous experiment of Fick and Wislicenus, known as the experiment of the ascent of the Faulhorn. It was shown by these observers that at least three times as much work was done during the ascent as could be accounted for by the oxidation of proteid, as estimated by the amount of nitrogen eliminated by them during and after the work.

"The work, therefore, could only have been caused by the oxidation of non-proteid matter. Similar results were obtained by Parkes and others in man, and by C. Voit in dogs. This, combined with the fact that the CO_2 output of the body is increased in proportion to the amount of exercise, led to the view being widely adopted that the energy of the body is mainly, if not entirely, obtained by oxidation of non-proteid materials, and that the splitting and oxidation of proteid must contribute, under the ordinary circumstances of a mixed diet, but little to the production of muscular energy."

Extract from the same book, p. 904 :—

"That the change in proteid which results in the formation of urea must *primarily* occur within the muscles, within which, as we have seen, the greater part of the oxidations of the body occur, there can be very little doubt. But there has always been this difficulty in connection with the question, that although urea is the ultimate product of proteid metabolism, the muscles practically contain either no urea, or only a very small amount."

With reference to these difficulties, I can speak from personal experience of the physiological effects of climbing mountains. For many years, after a sedentary life during eleven months of the year, I went during the month of August to Switzerland, or some other mountainous country, and walked as straight as possible across the country, taking the mountains as I

came to them. The most important physiological effect observed was naturally that of the excretions of the skin: the sweat from the face alone would leave a track as I walked. It will be observed that the excretion of the skin is not mentioned in the 'famous experiment' cited above, which is recorded in most of the physiological text-books.

Another important fact was that the temperature of the body was raised from 1° to 2° F., showing that very active chemical changes were taking place in the tissues. This fact is also left out of account.

The greater elimination of carbonic acid gas which naturally resulted from these ascents is mentioned, but the important fact that the urine was much diminished in amount, owing to the excessive sweating, was not noticed. After the first day there was no appearance of urates, nor any increase of colour to indicate that the urine was more concentrated. I did not examine the urine for nitrogen. The idea that the amount it contained was an index of the work accomplished appeared to me a preposterous proposition.

It is perfectly obvious that my muscles were throwing out a large quantity of lactic acid, and this would convert the urea into ammonia, and this ammonia would find its outlet by the skin and the lungs.

The enormous amount of nitrogen which is lost through the skin and the lungs in the form of ammonia has never been taken into account in any of these experiments. Yet this ammonia is very evident to the senses if we enter a barrack-room when soldiers have just come back from a march. Parkes, in his work on *Practical Hygiene* (p. 104), mentions this, and also notes that there was no augmentation in carbonic acid gas. Parkes also quotes some experiments made

by Moss on the air of a military hospital. He found that the quantity of ammonia in milligrammes per cubic metre was 0·855, and of albuminoid ammonia 1·307.

But the excretion of ammonia must not be regarded as only occurring after exercise. I find it very frequently present in the sweat of patients confined to bed, and it can at any moment and under all conditions be found in the air expired from the lung. It is only necessary to place a drop of hydrochloric acid on a glass slide, allow it to become nearly dry, and then breathe upon it (*see Plate VI, Fig.* 29). We shall at once obtain crystals of chloride of ammonium. Ammonia is exhaled in a free state from the skin. This can be demonstrated by taking some cotton material and soaking it in phenol-red; as it dries in the air, it will be turned yellow by the carbonic acid gas present. If this is placed at the bottom of a pill-box and the box attached to the skin so that the yellow cotton does not come in contact with the skin, the ammonia evolved will react on the colour of the cotton and turn it red. This effect is best observed during exercise, or when the patient is in a vapour bath.

But now we come to another riddle of physiology, which has led to the introduction of theories that have been the cause of much controversy. As urea is the ultimate product of the katabolism of protoplasm, why do we not find it in the muscles? If we adopt the theory that the energy of the muscles is due to non-proteid material, i.e., the carbohydrates, we have still to account for the dead cells of the muscles, which must contain proteid matter. There is no doubt that urea is not only formed in the muscles but must occur in large quantities.

PLATE VI.

Fig. 29.

Facing page 82.

The fact that the chemist fails to find it is no proof to the contrary. That the urea formed must come in contact with lactic acid is certain, and if so it would be converted into ammonia; but if this were the regular course we should produce a great deal more ammonia than we do, and man would become a very unsavoury animal.

The normal product of muscular contraction is carbonic acid gas. What effect would this have upon the urea?

Finding no information on the point, I made a solution of urea and passed a stream of carbonic acid gas through it. I then examined the solution under the microscope. I did not find urea; I did not find any combination of urea; I found *nothing*. Obviously the carbonic acid gas had broken up the urea into its ultimate elements—carbonic acid gas and nitrogen.

To look for urea in muscle is like seeking the paper with which the servant lighted the fire in the morning; and failing to find it, evolving a theory that she had discovered a way of lighting the fire without paper. What becomes of the nitrogen produced? The bulk of it unquestionably is got rid of by the skin, although some may find its way through the lungs.

It will be seen therefore that experiments to determine the amount of nitrogen eliminated which depend upon the quantity existing in the urine are entirely fallacious; practically the whole of the nitrogen evolved from muscular contraction is not taken into account.

But if we could measure all the free nitrogen, ammonia, and urea eliminated by the skin and the lungs during severe exercise, and add it to that obtained from the urine, I think the amount would not equal the energy

expended. I think so, because during my walks, which lasted from seven to ten days, the amount of food taken was not equivalent to the energy expended. There was always a marked absence of desire for food. A roll and butter at 6 a.m. and the usual continental sandwich during the day was all I found necessary. The usual supper was soup, omelette, and cheese. It was not a loss of appetite from over-fatigue, but rather from the sense of being fully satisfied, as one might feel soon after a good meal.

I have met with many others who on their mountain excursions had the same feelings, which they ascribed to 'living on the air'. But this explanation is not correct, because visitors at the high mountain resorts who spend most of their time out of doors do ample justice to the excellent fare provided, as I do myself during the periods of rest.

There can be no doubt that during great and prolonged physical exertion, also during fever, some products of metabolism, normally excreted, are used as a source of energy. This is altogether apart from nourishment derived from destructive changes in the tissue, as during starvation. It seems possible that the nitrogen set free in the muscles by the destruction of the urea is partially reabsorbed and utilized to supply the demand for nitrogen which exists under these circumstances. In no other way can I understand how a man in perfect health, and creating that demand by vigorous exercise, is satisfied without taking the extra amount of food regarded as theoretically necessary, and desires considerably less than would be taken by a person of sedentary habits.

Physiologists have never sufficiently recognized the skin as an organ of excretion. The sweat ducts have

been described as having a '*secretion*' of their own, chiefly consisting of salt and water, but it is recognized that urea and other products have been found in the sweat. The sebaceous glands have been described as secreting various fatty acids. The capillaries, by their dilatation, are known to liberate heat from the body and by their contraction to retain it, but they are not recognized as part of the machinery for oxidizing the waste products in the great lymph-space beneath them.

Functional inactivity of the oxidizing and excretory powers of the skin is the cause of many ailments the physician is called upon to treat—and fails to relieve because the cause is not recognized. If we watch the life history of those who suffer from this physiological deficiency, we shall find that eventually some form of nephritis gives expression to the condition, and the life of the patient is shortened. We try by diet to prevent the accumulation of 'uric acid' in the tissues, but take no steps to provide for its elimination by the skin.

Long clinical experience has led me to connect the persistent subnormal temperature with imperfect performance of the functions of the skin. There are doubtless other causes of a subnormal temperature; but when we find a patient in good health, and all the other functions performed properly, with a persistent subnormal temperature, we may suspect that the functions of the skin are in abeyance.

When the skin is excited to a large degree of activity, as during vigorous exercise, the temperature of the body rises above normal. I do not claim that the whole of the rise of temperature is produced by the oxidizing processes taking place in the skin, but it is obvious

that these processes could not be going on over the whole surface of the body without causing a rise of temperature. Therefore we must not regard the skin as a simple regulator of heat, but as a *source of heat*, and this point is of great clinical importance, because if we keep the skin warm we increase its functional activity, and this activity becomes a source of heat.

Before considering the matters excreted by the skin, it is important to remember that the act of sweating is of two distinct kinds. Visible fluid may be made to appear on the skin as a result of purely nervous influence, such as pain, emotion, or shock. We may produce copious sweating by the application of very high temperature to the nerves of the skin, such as would be produced in the hottest room of the Turkish bath. Sweating produced by such causes is not accompanied by any rise in the body temperature, the fluid excreted is chiefly salt and water, and although it may carry with it any matter which may exist in the ducts themselves, or in the lymph-space beneath, it does not set free the lactic acid which is locked up in the tissues of the body or in a state of combination. The other form of sweating represents a real increase in functional activity, rather than a paresis ; it occurs after vigorous exercise, as the effect of moderate degrees of heat, and when radiation is checked either by clothing or moisture in the air. This sweating is always accompanied by a rise of temperature of the body, and represents its normal function.

Both by physiologists and clinically these two forms of sweat production have been confused. They are physiologically distinct, and the resulting excretion has not the same chemical composition.

Schäfer (*Text-Book of Physiology*, p. 671) gives the chemical analyses of what he calls the '*secretion* of the skin', made by three different chemists.

In 1000 parts.	Favre.	Schottin.	Funke.
Water	995·573	977·40	988·40
Solids	4·427	22·60	11·60
Epithelium	—	—	—
Fat	·013	—	—
Lactates	·317	—	—
Sudorates	1·562	—	—
Extractives	·005	11·30	—
Urea	·440	—	1·55
Sodium chloride	2·230	3·60	—
Potassium chloride	·240	—	—
Sodium phosphate	Traces	1·31	—
Alkaline sulphates	·110	·39	—
Earthy phosphates	Traces	—	—
Total salts	—	7·00	4·36

It will be observed that there is a wide difference between the results obtained by different observers. This is inevitable, because there is no constancy in the composition of the sweat. It is seldom precisely the same in any two individuals; it may vary in the same individual at different periods of the day, according to physical conditions, and it may also vary in different parts of the body in the same individual at the same moment.

Chloride of sodium is always stated to be the chief component of the sweat. This is only true in chemical language. Crystals of pure chloride of sodium are met with very infrequently in the sweat. They usually occur at the end of very profuse sweating or in the sweat of paresis or of very high temperature.

Chloride of sodium is almost invariably found in

combination with other bodies. We have already discussed its combination with urea, and the crystals described are observed with great frequency in the sweat, and enable us to judge the amount of urea present. It is well to remember that the presence of the urea-sodium-chloride dagger crystals does not imply a very small proportion of urea if the minute urates are also present, as they usually are.

In the chapter on lactic acid I have described and illustrated the combination of sodium chloride with lactic acid. The form most frequently met with in the sweat is the one like a scaling-ladder with a number of lateral rods of unequal length. It has some resemblance to the lactochloride of ammonium. When any doubt occurs, it may be cleared up by adding a drop of Nessler's solution to the specimen, when, if the ammonia salt is present, an orange-coloured precipitate will be formed. I have never found chloride of ammonium except in combination with lactic acid.

The photograph of a drop of sweat (*Plate VII, Fig.* 30) shows urea in combination with chloride of sodium in the proportion of one to eight. At the lower part of the plate we see excellent crystals of lactochloride of ammonium. This also demonstrates that the urate was able to be formed because the lactic acid was locked up by the chloride of ammonium.

Only one of the chemists whose analyses we have quoted has apparently found lactic acid in the sweat, but it appears in so many forms that its entire absence is very rare. In addition to existing in combination with chloride of sodium and ammonium, it also appears as the lactophosphate of lime. This salt in its

PLATE VII.

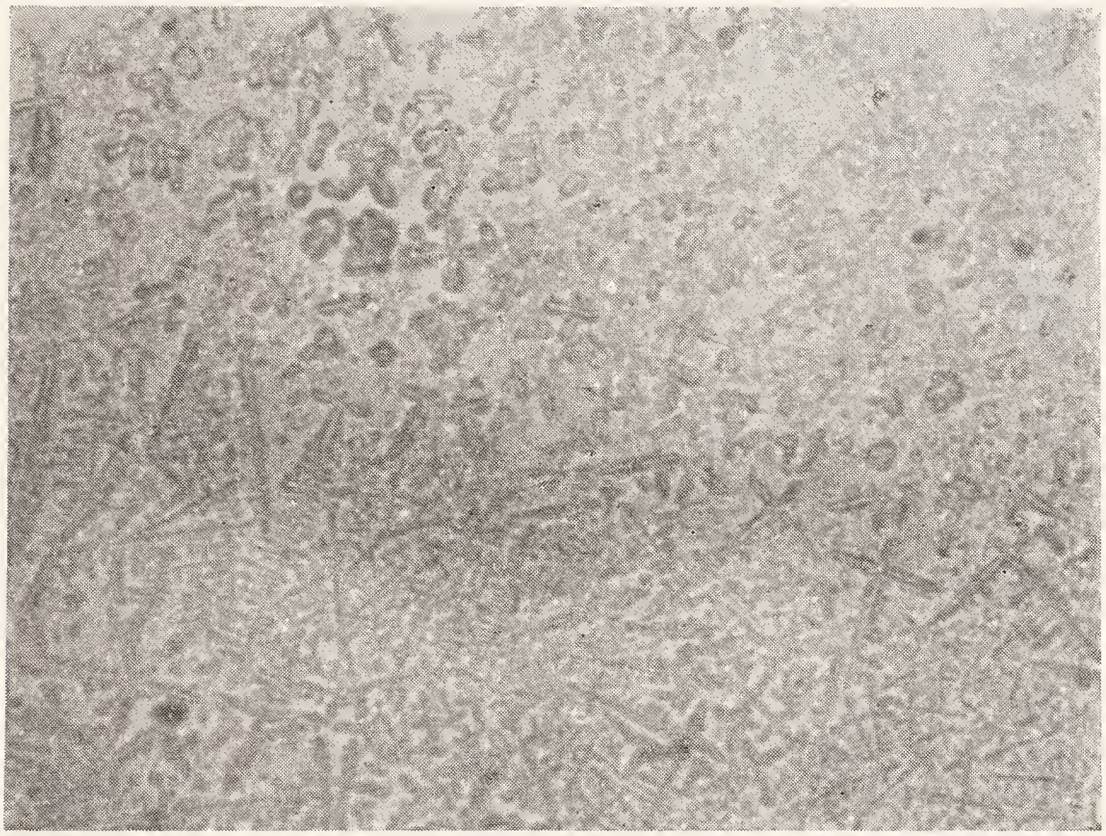


Fig. 30.

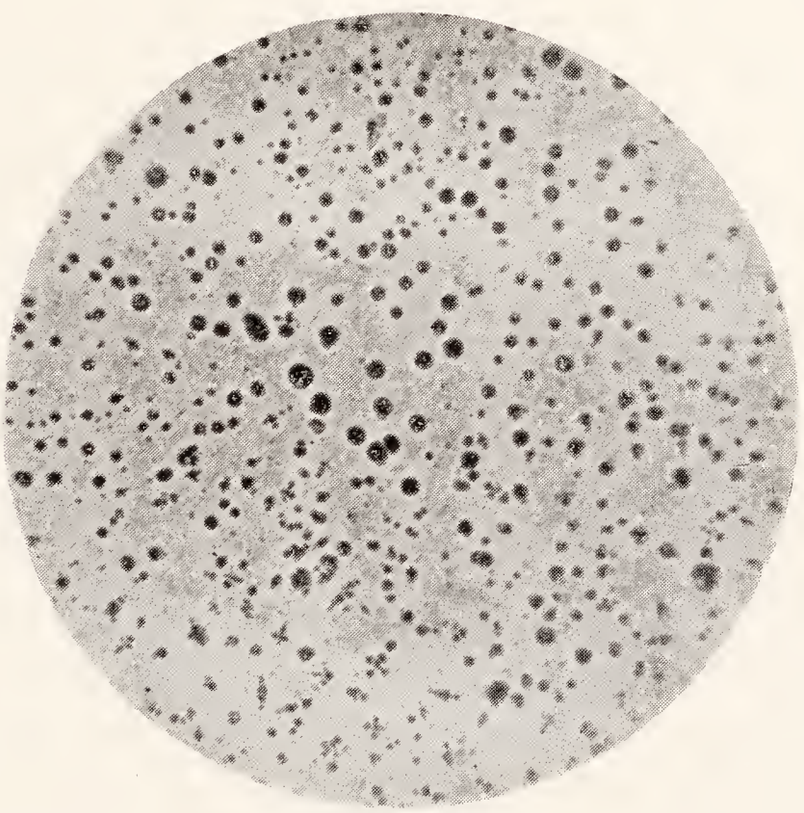


Fig. 31.

unoxidized form is usually found in large quantities in the sweat of rheumatic patients, although its existence appears to have passed unnoticed.

Fig. 31, *Plate VII,* is a negative of the sweat of a rheumatic patient, wholly consisting of minute urates, which do not appear in the photograph, and soluble lactophosphate of lime.

But there is still another combination of lactic acid found in the sweat, which also may exist in large quantities. This is the acid lactate of ammonium. I have already explained that when free lactic acid enters the lymph-space in large quantities it may combine with urea, because the urate cannot be formed in an acid solution. It converts it into lactate of urea, which passes rapidly into lactate of ammonium. I have never found a crystal of lactate of urea either in the sweat or the urine.

Lactate of ammonium has an alkaline reaction, and turns litmus paper blue in spite of the large quantity of lactic acid it contains, owing to the remarkable power which ammonia possesses of neutralizing lactic acid. Therefore an alkaline reaction to litmus paper is no proof of the non-existence of lactic acid. The difficulty this would otherwise present is diminished by the fact that it is nearly always present as an acid lactate of ammonium, and this, instead of turning litmus paper red, gives it a salmon-pink appearance, which is diagnostic of the presence of acid lactate of ammonium. But we meet with conditions when the sweat turns litmus paper neither red nor blue, but decolorizes it and leaves an almost white surface. This is due to a more highly oxidized form of lactate of ammonium, and represents a very large amount of lactic acid in the tissues and the lymph-space. These facts caused me

some trouble before I understood them, and their importance for diagnostic purposes is very great.

I have already alluded to the very large amount of ammonia which is excreted by the skin under certain conditions. It occurs in the sweat with such great frequency that it is remarkable that none of the analyses of the sweat contain any mention of it.

It is only by constant observation of a large number of specimens of sweat and under different conditions that we can really gain any information about it, and then we shall be impressed with the very wide variations which exist in its chemical constituents.

It would be impossible to make a quantitative analysis, and, if we did, the facts elicited are not very helpful; but the method I have described enables a dozen specimens of sweat to be examined and reported upon in the course of an hour, and the knowledge obtained is exact.

When we have done so, no doubt can be left in our minds that we are dealing, not with the secretion of a gland or a duct, but with the constituents of the great lymph-space which lies beneath the skin; and we can go further than that, and say we are observing the actual products formed in the tissues immediately beneath the point whence the sweat was taken. It may be a little difficult to believe this at first, but the following observations give it proof.

The reaction of the sweat has been described as both alkaline (Sir Michael Foster) and acid (Schäfer). Both observers may be right, because the reaction varies with the part of the skin whence the reaction is taken. The skin is sometimes neutral, rarely decidedly alkaline, more generally very slightly acid if taken over the chest wall. In the same person, at the same time,

the reaction of the skin of the palm of the hand may show some acidity, while the sole of the foot may be decidedly acid.

Except when there is some marked and general production of lactic acid, we never find the same degree of acidity over the whole skin. As a general rule the extremities, hands and feet, are more acid than the remainder of the body, and the feet more acid than the hands; but the forehead has usually a smaller degree of acidity than either.

Although there are differences in the reaction of the skin in different parts of the body, the records taken of patients who are under treatment present a certain uniformity.

The rheumatic patient has a very acid reaction at the outset, and this may continue for an indefinite time, according to the nature of the case. Then there comes a steady diminution in the degree of acidity, until the reaction becomes very slightly acid or neutral.

The patient with the lithic diathesis runs an entirely different course. His reactions are neutral or very slightly acid at the outset, and gradually increase in acidity until they reach a certain point, when they begin gradually to diminish in acidity until they are neutral or very slightly acid.

If we take fifty charts on which the skin reactions are recorded, it is quite easy to sort out those of rheumatic from those of gouty patients, without knowing anything about the patients or their symptoms. I know of no way which enables us so clearly to distinguish between the two classes of cases.

The fact that a patient with gout has had a very acid reaction and it has come down to neutral is no indication that the lithates have been completely removed;

it only means that we have got rid of all his additive lactic acid, and in the course of doing so have removed as much of the lithates as the acid set free was capable of doing.

It is better, therefore, to discontinue treatment and wait some months before renewing it, and this may be rendered unnecessary by the improvement that has taken place in the functions of the skin. In many cases the lithates subsequently disappear without further treatment.

I began making experiments on these points some twenty-five years ago, and I found them so helpful, both for purposes of diagnosis and as a guide to treatment, that I have continued them as a matter of routine ever since. As over a hundred such observations are made and recorded every day, I can speak on this subject with some authority. I could give many instances to show that the chemical composition of the sweat immediately over any part of the body is influenced by the processes taking place in the tissues beneath it; but one which any practitioner can observe for himself is, I think, conclusive.

If a piece of litmus paper is moistened and applied to a joint during an attack of acute gout, it will give an intensely acid reaction. If, now, the same experiment is made at a point on the skin a few inches away from the joint, the reaction may be only slightly acid. Personally I use for experimental purposes discs of cotton material which has been soaked in phenol-red and dried. In the process they turn yellow, and in this state are valuable for testing for ammonia, but for determining the acid reaction of the skin they are kept in a bottle containing ammonia, which restores the pink colour.

When moistened and placed on the skin, the time taken for the disc to turn yellow affords a very good indication of the degree of acidity.

I have occupied much of the reader's time in discussing various chemical and physiological problems, but I have tried to keep to the facts which have a direct clinical bearing upon the study and treatment of the rheumatic and gouty disorders. With the knowledge gained from these simple experiments we can approach the subject with a better understanding of the problems with which we have to deal, and work out the right line of treatment which the conditions present in the individual patient suggest.

CHAPTER VII.

RHEUMATISM AND THE LACTIC-ACID DIATHESIS.

The term rheumatism is applied to a painful condition of the joints or tissues which may exist with or without a rise of temperature. It is one of the symptoms of an underlying condition, but not a necessary one, for the state of the tissues which causes rheumatism may produce other symptoms, or no symptoms at all.

We associate rheumatism in its acute form with an exceedingly acid condition of the cutaneous excretion, and we know that the cause of this acidity is an excess of lactic acid. But an intense acidity of the skin is compatible with an appearance of perfect health; in fact we commonly find these symptoms in persons who appear to be exceptionally robust. If, however, we watch the life-history of such people, we shall have a much clearer understanding of the nature of the disorder, and shall observe that these persons are more liable than others to suffer from certain ailments.

An excessive acidity of the skin proves not only that the person is producing an excessive amount of lactic acid, but also that he is able to excrete it. This capacity for excretion may, under some conditions, cause trouble. We may find that the skin becomes raw and abraded from the action of the acid lying in contact with it, and this occurs most frequently where two portions of the skin are normally in close apposition. We call this eczema, and if we apply the usual ointments may find

it difficult to cure because we have not removed the cause of the condition. If we employ a calamine lotion or some alkaline powder or adopt any means by which the excretion is prevented from irritating the skin, we can relieve the local symptom although we have not removed the cause, but the success of such treatment will make the cause clear to us.

It must not be inferred from this that I trace all cases of eczema to an excessive acidity of the skin. On the contrary, some of the most inveterate cases I have seen are those in which the excretory powers of the skin are almost completely in abeyance. When, in such cases, we cause the functions of the skin to be resumed, we at first find neither urea nor lactic acid excreted, but I have found formic acid, which will account for the intense irritation of the skin generally observed in these cases. The sweat, when the excretion is first resumed, has the appearance of milk, owing to the enormous number of epithelial scales which are present.

This is a digression from my subject, but it may serve to call attention to the fact that the treatment of skin diseases without any attempt to study the condition of the cutaneous excretions and to remedy any defect which may exist in the performance of the functions of the skin must lead to failure.

Excessive acidity of the skin is found under two different conditions. There is an acid sweat which only appears as a result of active exercise or from the effect of the temperature to which the body is exposed, and there is also a passive excretion of acid sweat which may continue without the presence of any agency to excite it.

The first is due to the physiological activity of all the

factors concerned in the skin excretion, and removes the additive lactic acid from the tissues. The second is due to an excess of free lactic acid, stimulating the excretory functions of the skin, but depressing its circulatory activity and its power of oxidation. Such sweats do nothing to remove the additive lactic acid from the tissues, and so give no relief to any rheumatic or other symptoms which may be present. This form occurs in debilitated subjects. But both classes are subject to a common danger. Some portion of the body is exposed to what is called ' a chill '. It means that the nerves governing the circulation to the capillaries have received a shock which causes their continued contraction over the area affected. This condition may continue for days or weeks, most generally without the patient being conscious of it, but it can be determined by examination, and becomes more noticeable when the temperature of the body is raised, when the patient will complain of ' coldness ' in this particular part.

The first effect of the chill is that lactic acid is not excreted by the skin, and some portion of it combines with the cell-walls of the tissue. If there is a reaction, the patient may within twenty-four hours complain of tenderness over the part and pain on movement. We then connect the condition with the word 'rheumatism'. But the reaction does not always occur, or may be delayed for weeks, when some physical cause, such as a sudden rise of temperature of the body, may excite it ; or under favourable circumstances the whole condition may slowly return to normal without the patient being conscious of what has taken place.

But a chill may be general and involve the whole body. This checks the excretory action of the skin, and a large amount of lactic acid finds its way to the

cell-walls of the tissues. The symptoms presented will depend on whether a reaction takes place or not. In the absence of any reaction, the patient will complain of feeling muscularly tired; he will have all the secondary symptoms of over-fatigue without having done anything to occasion it. There will be indisposition to exertion, but if he should be forced to take exercise, he will feel rather better instead of worse for it. This distinguishes it from true debility. There is often great mental depression, and in severe cases it may almost amount to melancholia. Such cases are met with frequently in practice, and are usually treated as 'neurasthenia'. Unless something happens to set up a reaction, this condition may continue for an indefinite period.

Of course there is a marked variation in the degree to which patients suffer from these symptoms. We may have anything from a general indisposition to exertion to a profound depression ending in suicide. Reaction may take place within twenty-four hours of the chill, or be delayed for weeks or months, when some agency other than the original chill sets it up. It may be an ordinary 'cold', exposure to heat, or an attack of influenza. Any condition which raises the body temperature may produce the reaction.

Lactic acid may be regarded as matter in a state of incomplete combustion, and once rapid oxidation takes place, as during fever, a high temperature results. Given an excess of lactic acid in the tissues, we are likely to have a high temperature, and usually accompanied by very free cutaneous excretion of a very acid character. Such cases may run a course of a few days or a few weeks, the only symptoms present being the temperature and the sweating. The more moderate

both symptoms are, the longer the condition may last. Such cases are usually diagnosed as 'influenza', but when they continue for a long time the diagnosis may be changed to 'typhoid', and sometimes they out-run the temperature duration of this diagnosis. I have been called in consultation to cases of this kind which have puzzled the physicians attending.

There is one symptom which I have observed in almost every case: the pulse is not as rapid as the temperature would lead one to expect, and is much slower than in a case where inflammation exists. There is also an intensely acid reaction of the skin. The explanation of these cases becomes very simple when the symptoms are accompanied by swelling and inflammation of the joints. Then no one is in doubt as to the nature of the diagnosis—it is recognized as acute rheumatism. We see then that a healthy man who has an excessive production of lactic acid is liable to a number of conditions all due to the same cause, and which receive different names because the symptoms differ.

Many years ago I called attention to the importance of the reaction of the skin as a diagnostic symptom. A physician present at the meeting sent for some blue litmus paper and tested his own skin. The reaction was intensely acid. He then produced the paper and stated that he had never been in better health in his life. He was under the impression that this was a conclusive argument against my view Some years later, I found myself seated next this physician at a dinner, and he told me that he had been out of practice for over twelve months from a severe attack of 'rheumatic gout'. Clinical evidence in respect to rheumatism and gout can only be based on the life-history of cases,

because the removal of symptoms is no evidence of cure, and the accession of symptoms or the aggravation of those existing during the attacks is often the best evidence that good results are being obtained. It is the condition of the patient months or years later that gives us the only evidence we can rely upon.

Having diagnosed the case as acute rheumatism, because the fever, the joint symptoms, and the acidity of the skin clearly point to it, we shall regard this as a reaction designed to relieve the body of the acid which saturates the tissues. But we are taught not to take this view. On referring to Sajous' *Encyclopædia of Medicine*, I find (p. 479) that "the conception of the nature and origin of the disease has, however, completely changed during the last decennium. It is now commonly considered an infectious disease. This view is based mainly upon the fact that rheumatic fever is an epidemic disease, and that during epidemics the cases accumulate in some houses, whereas other houses are quite spared. . . . . Although it is commonly admitted that rheumatic fever is caused by an infectious micro-organism, it has not yet been possible to discover the specific microbe".

The only claim advanced to prove rheumatic fever an infectious disease is that of clinical experience, and this experience is one of epidemics of rheumatic fever. For thirty years I have been examining patients from all parts of Great Britain who have had attacks of rheumatic fever, but I have never yet met with one who contracted the fever during an epidemic of the disease. I have never had any personal opportunity of observing an epidemic of rheumatic fever, and I am not, therefore, prepared to assert that such a thing is impossible. On the contrary, just as at certain seasons

of the year and under certain climatic conditions we have epidemics of influenza, so at a particular season of each year we have the largest number of cases of acute rheumatism. This season is when the damp and cold of winter give place to the more genial atmosphere of spring. The temperature of the air rises rapidly, and at this time acute rheumatic complaints become more prevalent, so that we find that April, May, and June are the months when we expect to see most such cases. This is clearly not due to infection, but because during the cold months of the year the excretion of lactic acid is less active, and a large quantity is locked up in the tissues, and a sudden rise of the temperature of the air tends to set it free and produce symptoms.

Another possible exciting cause is the bacillus of influenza. In respect to this we know that periodically we have an infectious disorder prevalent. It is not a new thing, because Hippocrates described it some centuries before the Christian era, and attributed the disorder to climatic influences. We know that each epidemic has its own characteristic symptoms, and these may be of a very varied character. We have had epidemics when the symptoms nearly approached those of dengue fever, others when pneumonia has been a frequent result, also epidemics causing temporary nephritis, others where eruptions resembling scarlatina and measles were marked symptoms; but more generally we have cases where the upper air-passages are affected and pyrexia is mild or absent. We must infer from this that either there are different 'influenza' bacilli, or that new bacilli of a mixed character are created, or that the influenza bacillus has the power of breaking down the resistance to other bacilli which may exist in the organism at the moment

of the attack. These are questions which must be answered by the bacteriologist. Clinically, we have the fact that if we examine swabs from the throats of a number of healthy persons, we may find the diphtheria bacillus, the pneumococcus, *Bacillus coli*, streptococcus, staphylococcus, etc. Each one of them is capable of producing symptoms more or less serious, but fails to do so because of the resistance offered by the organism.

In dealing with cases of the lactic-acid diathesis these points are very important. One of the most common symptoms met with in such cases is a mild chronic pharyngitis, with some elevation of the papillæ and slight congestion. This symptom passes unnoticed, and the only symptom produced is the tendency to hawk up mucus, and 'clear the throat'. In cases where there is great debility we may find a membrane over the back of the pharynx which closely resembles that of diphtheria. It occasions no alarm because it is not accompanied by any constitutional disturbance. But patients with the lactic-acid diathesis are very liable to acute inflammatory affections of the tonsils, accompanied by a high temperature. The attack may take the form of follicular tonsillitis, and in this case its diagnosis presents no difficulty. If, however, the practitioner is anxious to avoid any mistake, and takes a swab and sends it to a bacteriological laboratory, he will very likely be informed that the diphtheria bacillus is present and the case is one of diphtheria. He will therefore feel bound at once to administer antitoxin, although all the clinical symptoms are those of follicular tonsillitis. But when the tissues are charged with lactic acid they are very intolerant to antitoxin, and in such cases it is likely to cause serious irritation of the kidney or inflammatory affections of the skin.

These cases clear up rapidly when a solution of biniodide of mercury is applied to the tonsils and pharynx. I use 1 gr. of the biniodide to 4 oz. of water, with a few grains of potassium iodide added to effect solution. A small quantity of this is sprayed or painted on the tonsils every three hours. Immediately before the application is made I add 10 drops of peroxide of hydrogen to the portion of the solution to be used. This causes decomposition, and the solution is used in a nascent form which, I think, adds to its efficiency.

The whole question has been complicated by the insistence on the view that rheumatic fever is itself an infectious disease. Its relation to inflammatory affections of the tonsils has led to the theory that it is by the tonsils and pharynx that it gains admission to the body. If we accept this view, we must suppose that a healthy man is liable to become infected with rheumatism, and that rheumatic fever is the expression and natural result of that infection. We cannot explain how he becomes infected. The rheumatic-fever patient has never been known to infect any other person. This difficulty has been got over by explaining that the bacilli are too deep in the joints to infect other persons.

Such arguments betray a great lack of knowledge of the clinical symptoms of acute rheumatism. Beyond all other diseases, rheumatic fever is a disorder attended by the excretion of the poison which causes the disease. When that excretion is finished—and we know when by the return of the reaction of the skin to normal—all the symptoms subside and the patient recovers. The bacilli cannot remain deep down in the joints under such conditions.

If we examine the history of any patient previous to the attack of rheumatic fever, we shall invariably

obtain evidence that for weeks before the attack he suffered from some of the symptoms I have already described. Many years ago I gave the name 'asthenoxia' to this condition. It may not prevent a man doing his work, but he does it as a tired man, and keeps going by effort. When rheumatic fever is properly treated, the malaise is the first symptom to disappear.

The bacteriological theory of rheumatic fever is wholly unnecessary to explain any single symptom of the disease, and raises questions which it is impossible to answer. It causes confusion of thought on a matter which, in every detail, is otherwise simple and intelligible. But just as we have sporadic cases of diphtheria and diphtheria as an infectious disease, so it is alleged that we have epidemics of rheumatic fever and that it assumes an infective form. I have no personal knowledge of such epidemics, but I can easily imagine the conditions under which they could occur.

Take a community who earn their living by hard physical exertion, involving strain upon the joints and tendons. We have only to suppose a period of cold and damp weather when the excretion of the skin is inactive, followed by a mild attack of influenza which raises the temperature of the body, for all the symptoms of rheumatic fever to be set up in a considerable number of people at the same moment.

If the bacillus happened to be that of scarlatina, the result would be the same. Any bacillus, or any physical cause which would suddenly raise the temperature of the body under such conditions, would be sufficient to set up either rheumatic fever or a high temperature without joint symptoms. As a matter of fact we see such cases during every epidemic of influenza; but if

the joint symptoms appear we call it rheumatic fever, and if there are no joint symptoms we regard it as a severe attack of influenza. But other persons in the same house, infected by the same bacillus, will show a very slight rise of temperature, which may subside in twenty-four to thirty-six hours. No clearer proof could be given that the patient who has the high temperature, lasting perhaps for two or three weeks or longer, has some underlying condition which makes the symptoms he presents altogether different from those exhibited by other patients.

It is well known that some of these cases make a rapid convalescence after an attack of high fever; others may remain debilitated for months afterwards. It is not difficult to give an accurate prognosis as to which result is likely to happen. If we take the reaction of the skin during the attack, we shall find that it is extremely acid. This is invariably the case unless the excretion of the skin is arrested, and this rarely occurs. If we watch the reaction of the skin, and find that it remains intensely acid for some days after the temperature has fallen, we can predict a prolonged convalescence. If the acid reaction of the skin diminishes as the temperature falls, we can say with certainty that the patient will make a rapid recovery. It is many years since I first described this prognostic symptom, and I have never known it to fail.

But when the acid condition remains after the temperature has fallen, I do not give an unfavourable prognosis, because I take immediate steps to carry on the work which nature has failed to accomplish. I will explain this method in the chapter on 'Pyretic Treatment'. The point I am anxious to insist upon

is that in most cases where there is prolonged convalescence from 'influenza', there exists an excess of lactic acid in the cell-walls of the patient's tissues, and if proper means are taken to remove this, they will ensure a rapid recovery. It is for this reason that tonic remedies fail in such cases. The cause is practically physical, and only physical methods can assure good results.

I will deal with the symptoms of chronic rheumatism in the chapter on 'Arthritis', not because the term should ever be applied to it, but because it will be a more convenient place to discuss its differential diagnosis from 'rheumatoid arthritis', with which it is commonly confused.

CHAPTER VIII.

RHEUMATIC FEVER.

In a typical case of rheumatic fever there are practically three distinct symptoms: (1) The fever; (2) The inflammation of the joints; (3) The acid excretion of the skin.

We see a large number of cases where only the second symptom exists. There is an inflammatory condition of the joints, accompanied by pain, but there is no fever and no excessive excretion by the skin; this, in fact, may be altogether arrested. We know that in rheumatic fever, within a certain number of days or weeks the attack will come to a termination, under any kind of treatment; that in the case unattended by fever it may go on for years and increase rather than decrease in severity. This teaches us to regard the fever and acid sweats as symptoms of active metabolism rather than as symptoms of disease. It is because this lesson has never been learned that a clinical experience has been created which conveys the idea that rheumatic fever is a disease in which relapse is very frequent, convalescence is prolonged, and over 70 per cent of cases are left with valvular disease of the heart.

The physiological studies which I have recorded enable us to understand clearly why these results happen, and the method by which they can be avoided. We know that every patient who suffers from rheumatic fever has, prior to the attack, a large amount of additive lactic acid in the cell-walls. We know from our experi-

ments that the tissues are capable of taking up a large amount of lactic acid beyond that which is necessary to combine with the phosphate of lime in the cell-walls, and this addition makes no difference to the chemical combination of lactophosphate of lime, nor does it alter its crystalline form; but the additive lactic acid is in such close cohesion, that it is uninfluenced by, nor does it influence, alkaline fluids surrounding it. But when a rise of temperature takes place, some portion of the additive lactic acid is set free, and the process continues during the whole period of the fever. We know, therefore, that the patient has been exposed to certain conditions which either increase the production of lactic acid or have checked its elimination.

As regards increased production, we know that there are three factors concerned, acting either in conjunction or singly: (1) Increased muscular action; (2) Exhaustion of the nerve-supply; (3) Imperfect oxidation, from alteration in the quantity or quality of the blood-supply. It is important to determine these factors in every case, with a view to placing the patient under such conditions as will prevent this excessive production.

As regards the defect in its elimination, we have to consider the causes which check the normal functional activity of the skin or which prevent the oxidation of lactic acid in the tissues. Exposure to cold and damp are common factors in producing this condition, and deficiency of hæmoglobin in the blood is another factor found in a certain proportion of cases. There is no evidence to show that lactic acid is destructive to the hæmoglobin; but the absence of hæmoglobin is favourable to the presence of an excess of lactic acid.

We have already dealt with the symptoms which may occur, due to this condition, prior to the attack

of rheumatic fever. The immediate cause of the actual attack is any factor which may bring about a sudden rise of the temperature of the body. It may be a reaction from a chill, the presence of any bacillus, or the result of any physical condition which causes a temporary pyrexia. In my experiments with animal tissues saturated with lactic acid, I have shown that a rise of temperature liberates the additive lactic acid. I have further demonstrated that the presence of free lactic acid prevents urea from combining with chloride of sodium to form the urate, that lactic acid combines instead with the urea, to form lactate of urea, and this passes quickly into lactate of ammonia. In this way the alkalinity of the tissues is preserved.

But lactic acid cannot combine with ammonia without the evolution of heat. If we add lactic acid drop by drop to a solution of ammonia, we can maintain the temperature of the mixture for an indefinite period. When the tissues are saturated with lactic acid, as they are at the moment of the attack of rheumatic fever, the quantity of lactic acid liberated by a small rise of temperature will be sufficient to raise the body temperature to a still higher level, and this in turn will liberate more lactic acid, so that a further rise of temperature takes place; and this will continue until a balance is established between the rate of liberation of the lactic acid and the temperature reached, because as soon as the excess of additive lactic acid is removed, the rate of liberation is diminished. This will explain how it happens that the temperature in rheumatic fever almost invariably rises by stages to a certain level, and is then maintained at that level with small fluctuations over an indefinite period.

The chemical nature of the fever is indicated by the

fact that the pulse-rate is not under normal conditions increased to the same amount as would happen if the fever were the result of inflammation. When a rapid pulse occurs under such conditions, we can be sure that the heart is being irritated by the failure of the organism to neutralize the lactic acid. This result is most likely to occur if the normal functions of the body are interfered with by the ill-advised use of certain drugs. The whole treatment of rheumatic fever must be based upon the point of view from which we regard the temperature.

That fever is a pathological symptom has been taught by the physicians of all ages. Even at the present day the chief use made of the thermometer is to detect fever as a pathological symptom. I have found physicians who could not understand the reason for taking the temperature of patients two or three times a day when no fever existed. The enormous importance of the subnormal temperature has never been fully realized, because nurses do not always shake the index of the thermometer to 95° before they take a patient's temperature, and many temperature charts do not afford any space for these low temperatures.

In the treatment of all disorders of metabolism, the temperature, whether it is high or low, affords us the best chemical indication we can have of the chemical activities of the body.

We must begin by discarding the idea that a rise of temperature above normal is of necessity a pathological symptom. This is easily demonstrated by the fact that no healthy person can take active exercise without producing a rise of temperature. We know that exercise increases the production of lactic acid. It is probable that the rise of temperature of the body is in large part due to the excess of lactic acid produced

during exercise: even healthy people have some additive lactic acid in the cell-walls, and this is liberated during exercise. I think this conclusion is correct, because the man who takes active exercise daily does not develop the same amount of heat as a result of any specific act of exertion as the man of sedentary habits who takes active exercise occasionally. We may account for this by the fact that the man of sedentary habits has a larger amount of additive lactic acid in his cell-walls.

I have found some physicians so impressed by the heat-regulating mechanism of the body that they have doubted whether exercise does produce a rise of temperature. A medical friend disputed this point one night. The next day he met me and admitted that I was right. He told me that he had used the mowing-machine on his lawn, with a thermometer in his mouth. The heat-regulating mechanism of the body would be more effective if we did not happen to wear clothes.

The fever of rheumatic fever is as much a physiological process as the fever due to active exercise, and is due to precisely the same chemical activities. It was the study of rheumatic fever which first led me to understand that fever may be a normal physiological process, and also a very powerful therapeutic agent. When I brought forward this view at a medical society some twenty years ago, almost all my auditors thought it necessary to give their reasons for dissenting from this proposition. But within a few months, without any further arguments from myself, many of these, on reflection, came to the conclusion that the view was sound. At the time I was not in a position to arrive at my conclusions by induction, and had to depend upon deduction. Whichever method we employ the result is the same.

The view that the fever was a pathological symptom led to methods of treatment that would most quickly cure the fever, and the discovery of salicylic acid and its salts was hailed with great enthusiasm. It was necessary to give the drug in large doses to produce the result. Under its influence the fever subsided and the pain and inflammation of the joints was relieved, so that the patient was comparatively quickly able to be removed from the hospital. But from the Reports of St. Bartholomew's Hospital (Dr. Samuel West, *Practitioner,* 1888, p. 104), 70 to 86 per cent of the patients treated developed heart disease as a result of the attack.

Not only is heart disease regarded as an after-result from which few escape, but also prolonged convalescence and relapse are regarded as essential clinical symptoms of the disease. Thirty years' experience in the treatment of rheumatic fever and its after-results enables me to state positively that these clinical results are due almost in their entirety to a false conception of the nature of the disease and to the efforts to suppress the normal physiological reactions intended to avert these dangers. If rheumatic fever is not interfered with by drugs or any agents, it will run its course in a certain number of weeks, and both the fever and the joint symptoms will disappear. In a case of chronic rheumatism we have the same joint symptoms, but fever is absent, and the condition may continue for years, and there is no tendency to natural recovery. It might be thought that these clinical facts alone would have made it clear that the arrest of the fever in rheumatic fever might have dangerous consequences ; but in spite of the very serious results which have followed the use of salicylates and similar remedies, they have continued to be employed.

The real problem in the cure of rheumatism is to find some agent which will liberate the additive lactic acid from the cell-wall. In my experiments with animal tissues containing an excess of lactic acid, it was shown that, if we surrounded the tissue with an alkaline solution, no chemical combination took place except when we added sufficient heat to liberate the lactic acid. This is the method of Nature, and I have discovered no other.

From this we might suppose that if we left patients without any treatment in rheumatic fever they would make a good recovery. Unquestionably they would be much better than with the results of salicylate treatment ; but during my long experience the number of patients I have seen where Nature brought the case to a satisfactory conclusion is very limited.

One case occurred in my early experience. A robust girl, 18 years of age, developed rheumatic fever. She had a very high temperature, all the joints of the limbs were involved, there was profuse acid sweating, continued acid vomiting, and a diarrhœa which consisted chiefly of a large amount of acid fluid. In this case I merely gave a placebo ; no help appeared necessary. The patient made a good recovery in three weeks, and has remained without any further attack for thirty years. The patient was of the well-marked lactic-acid diathesis, and her mother and grandmother both had valvular disease of the heart as a result of rheumatic fever.

In a number of cases the physiological reactions are sufficient in the early stages, and only need help if the skin should be inactive, as sometimes happens ; but there comes a time when the temperature falls and the patient appears to be approaching recovery. This is the most critical stage in the treatment of

rheumatic fever. If, in spite of the fall of temperature, the reaction of the skin remains very acid, even if the joint symptoms have subsided, we can be sure that the patient will have a prolonged convalescence and is in danger of relapse, unless we take artificial means to maintain the temperature and ensure the physiological activity of the skin. The reaction of the skin is the one clinical fact which must guide us in these cases. If we take means to prolong the attack until the skin becomes only moderately acid, we can then be sure that the patient will have a quick convalescence and be in no danger of relapse. The method of treatment adopted will be best considered in the next chapter.

The results of treatment of rheumatic fever when properly carried out are wholly satisfactory. Heart disease rarely occurs if the patient is seen at an early stage, and a blowing murmur frequently heard over the heart on admission usually disappears in the course of treatment. It is quite common for a patient with rheumatic fever, before the temperature and the joint symptoms have subsided, to express himself as feeling better than he has felt for months. The appearance of the patient affords evidence of this. The complexion, which had the dull opaque appearance which is significant of lactic-acid excess, changes, and there is greater translucency of the skin. The eyes also have a brighter and clearer appearance. These symptoms afford proof that the fever has removed something from the tissues which was depressing their normal physiological activity.

Relapse may be said never to occur in cases efficiently and *completely* treated. I emphasize the word 'completely', because the symptoms of 'cure' are often misleading. Thus, a healthy-looking boy appeared at

the out-patient department complaining of rheumatic pains in the muscles of the back. He had been sleeping on the ground in a boy's camp. Now, as a number of boys were exposed to the same conditions, there must have been some antecedent condition existing in the boy, as he was the only one who developed any rheumatic symptoms. His skin showed an extremely acid reaction. I attach very great importance to this symptom in children. The lactic-acid diathesis may, in such cases, first make its presence known by some permanent mischief to the valves of the heart, by pericarditis, endocarditis, or pleurisy. We may, of course, have far less urgent symptoms as a result of the condition; we may have pains about the body which used to be explained as 'growing pains'. Because I took the view that this symptom should be treated seriously, I ordered the boy into hospital, and gave him treatment which consisted in causing an artificial rise of temperature each day. On the third day he developed an attack of rheumatic fever, which ran an ordinary course for three weeks and then subsided. The boy expressed himself as quite well, and there was nothing to prevent his discharge except that the reaction of the skin was almost as acid as on the day of admission. I therefore ordered the treatment to be continued. Within forty-eight hours the temperature rose again and several joints were inflamed. This continued for a fortnight, when the attack subsided. His skin then showed very slight acidity and I dismissed him. I had the opportunity of watching the history of the case for ten years, and there was no return of the slightest rheumatic symptom. He developed into a very healthy man. In this case, if I had dismissed the boy when the symptoms were 'cured', he would

most certainly have had a relapse sooner or later, and the relapse would have been explained as the normal character of the disease. It would have been really due to the fact that my treatment had not been *complete*.

This case emphasizes not only this point, but also the fact that even in a healthy boy Nature may fail to complete the reaction, and, as a matter of fact, does fail to do so in the large majority of cases of rheumatic fever. It is on this account that, while we regard the symptoms as physiological which it would be wrong to suppress, almost every case calls for active treatment, and more especially in its later stages.

A large number of cases are sent to us as cases of rheumatic fever where, after prolonged convalescence, the symptoms of rheumatism in its more chronic form have developed. In these cases the treatment may reproduce the rheumatism in its acute form, and there may be a continued temperature for some time. When this occurs, recovery occurs at an earlier date than when a rise of temperature only occurs during the actual process of treatment. In either case the results are always satisfactory.

Some years ago a special number of the *Practitioner* was published in which physicians were invited to give their experience in the treatment of rheumatism. I remember reading of a case treated at the Glasgow Royal Infirmary by 'Wilde's treatment'. The hot blanket pack appears to have been used for raising the patient's temperature. Under this treatment the patient's temperature rose, and fresh joints became implicated. As this continued, a consultation was called, and it was decided to abandon the treatment. Other methods were employed, and the patient was

finally dismissed unimproved. Some three months later the man reappeared at the hospital quite well. On being asked what he had done, he said that he knew the treatment they started with was doing him good, so when he went home he got his wife to continue it. This case points to the necessity of the physician who conducts such treatment having the courage of his convictions, and always remembering that neither the fever nor the joint symptoms are pathological.

Another misfortune happens to rheumatic-fever patients because the fever is wrongly regarded. It is quite customary to order a milk diet to such patients. As all the tissues are saturated with lactic acid, and lactic acid is being excreted into the stomach and intestines, milk happens to be the one food that is likely to augment the existing conditions. As a consequence, a thickly-coated tongue with gastric disturbance is regarded as a normal symptom of rheumatic fever. With ordinary diet, with the exclusion of milk except in small quantities, and with the use of simple salines to assist the excretion of the intestinal contents, these symptoms do not occur.

We may divide rheumatic-fever cases into sthenic and asthenic. In the sthenic cases with high temperature, small doses of acetylsalicylic acid at infrequent intervals will modify the temperature and ease the pain, and also promote the action of the skin. There is no danger in the use of this drug or the salicylates so long as they are not used in antipyretic doses. In the asthenic cases, means to raise the body temperature should be employed from the first, and as in these cases there is usually defective oxidation, the use of nitrogenous foods is indicated and assists the treatment.

CHAPTER IX.

PYRETIC TREATMENT.

This term applies to any method which has for its object the elevation of the temperature of the body for purposes of treatment. It does not include processes in which hot dry air is applied to the body either generally or locally, or those in which the aim is to cause sweating, without regard to the temperature of the body. Before I discuss the technique of this method, it will be better to point out its therapeutic value and the conditions indicating it.

It was the study of rheumatism in its acute and chronic forms which first directed my attention to the value of fever as a therapeutic agent. Rheumatism attended with fever runs a definite course, and its natural tendency is to recovery. Rheumatism without fever has no tendency to natural recovery, and may go on for years and become progressively worse. As I have shown, acute rheumatism may drift into the chronic condition as a result of artificially suppressing the fever, or we may have heart complication and relapse from a similar cause.

The value of fever in the cure of rheumatism is demonstrable by the experiments I have described in the chapter on lactic acid. When we supersaturate an animal tissue with lactic acid, and wash and dry the specimen, we have a tissue that will present a very acid reaction to blue litmus paper, but which, if placed in an alkaline solution, will neither impart its acidity to

the alkali nor become neutralized by the alkali in the solution. I have demonstrated that this is not due to the whole of the lactic acid being in chemical combination; the bulk of it is simply 'additive' and held in a state of cohesion. I have shown that this cohesion can be overcome by raising the temperature of the fluid in which the acid tissue is placed, and that under these circumstances the acid is slowly set free. This explains why alkaline treatment of rheumatism, even if it relieves symptoms by combining with any free acid which may be present, will fail to cure, because it cannot liberate the acid in cohesion with the tissues. The production of fever is the method adopted by Nature, and so far I have not been able to discover any other method which will accomplish the same result.

There are many ways in which the temperature of the body can be artificially raised. It is not dependent upon any particular form of apparatus, although I have designed some as a matter of efficiency and convenience. In 1893 I published the results of five years' use of pyretic treatment (*Rheumatism*, Bale & Sons), and described a simple appliance I had designed for use in rheumatic fever. This was at once 'improved' upon for commercial purposes, and this in turn was still further improved upon by other inventors, whose aim appeared to be to submit the body to very high temperatures, and these appliances have been used all over the world. The result has been that the true object of the appliance I used has been completely misunderstood, and a method of treatment adopted which has many grave disadvantages. It is for this reason that it is necessary for me to trouble the reader with some elementary physiological facts.

The human body is a heat-producing machine, and

radiates heat. To apply a very high temperature to the human body we must employ dry air. If we used water or steam, we should burn the skin at a much lower temperature, because the vehicle we employed would check the heat radiation from the body. Hot dry air does not do so because it assists the radiation of heat. Thus the nude man in the hottest room of a Turkish bath will have a normal temperature. I say nothing about the dilatation of the blood-vessels favouring the radiation of heat, because this takes place with all forms of heat.

In order to raise the body temperature, we are only concerned with methods which check the radiation of heat from the body, and those which increase its production. But if we check the radiation of heat from the body and raise the temperature, without securing dilatation of the cutaneous blood-vessels and the consequent action of the sweat-glands, we induce a condition of congestion, which will be followed by malaise, because we have set free acid from the tissues without securing its elimination. Thus, if we take a man with dry inactive skin, and wrap him in a number of blankets, we can secure a rise of temperature of the body ; but if no action of the skin takes place, he suffers from congestion, which will be only relieved as the acid set free is eliminated by the lymphatics.

For this reason, when we raise the body temperature, it is necessary to use such a degree of heat as will dilate the blood-vessels and secure cutaneous excretion. It is at this point that confusion of thought occurs. It appears to many that any method which secures free cutaneous excretion is sufficient, and that this is the mode of treatment indicated. But we can obtain free excretion of the skin by hot dry air, as in the Turkish

bath, without raising the body temperature or setting free the lactic acid. It must be remembered that to obtain curative results by this method it is necessary to repeat the process daily perhaps for a number of weeks, and, if we over-stimulate the skin by heat, we shall exhaust the patient and also depress the activity of the skin. Some of the most difficult cases I have met with as regards restoring the functions of the skin have been patients who have had prolonged courses of Turkish baths, using the hottest room, or patients who had lived for many years in hot dry climates. It must be remembered that Turkish baths originated, and are most used, in hot climates where the skin acts too copiously, and the desired effect of these baths is to limit the overaction of the skin.

For purposes of treatment we need the lowest temperature which will accomplish the result, and applied through a vehicle which will check heat radiation from the skin.

In experiments, made many years ago, I found that I could obtain the maximum rise of body temperature with the minimum of heat by using moist warm air at 103° to 105° F. This was in patients whose temperature was subnormal or who had inactive skins. When the temperature is above normal, or the skin is active, a lower temperature is desirable, because, if we produce free action of the skin too quickly, the temperature does not rise to the same degree. We *diminish* the rise in the body temperature by *increasing* the external heat employed. This is a fact which the uneducated find it difficult to understand.

The ordinary immersion bath will raise the body temperature, but water in contact with the skin checks cutaneous excretion, and, unless this occurs after the

patient leaves the bath, is apt to cause the feelings of congestion I have described.

Hot baths do not greatly influence the excretion of lactic acid. We have evidence of this in the fact that they do not remove from the skin the dead epithelial cells, which requires lactic acid to overcome their cohesion to the living cells, as I have explained in a previous chapter.

Steam and vapour baths raise the body temperature, and the cutaneous action is only to a small degree checked by the condensation of the vapour on the skin. It is not an ideal method, but efficient enough in many cases if it can be used with the patient in the horizontal position. As steam baths dilate the blood-vessels, the sitting position is undesirable. The crudest form of bath I have seen is an electric bath in which the patient sits on a stool with his head through a hole in a box, surrounded by electric lights which give a perfectly dry heat. This is the antithesis of pyretic treatment.

In my own practice, where forty or fifty treatments have to be given daily, it is necessary to have some convenient appliance which enables the maximum efficiency to be attained with the minimum of labour. Many years ago, I designed a 'thermal couch' which has met every requirement. It consists of an iron bedstead, the mattress of which is a double layer of fine-mesh galvanized netting. On this are placed six blankets and a bath sheet. The patient lies upon these, with a pillow, as if in bed. A hinged metal cover is placed over him, covering the feet and reaching to the shoulders; the opening here is closed by a blanket, so that only the head is left uncovered. Beneath the wire mattress is a copper receptacle covering the whole bed, and closed at the bottom and sides. The sides

slope to the centre to allow the water of condensation to run away to a small pipe which acts as a drain. Into this chamber steam is ejected from a self-filling boiler. The steam heats the blankets upon which the patient lies, and finding its way through them, deposits all superfluous moisture, and fills the chamber in which the patient lies with warm moist air. This appliance produces a rise of temperature of 3° to 4° F.

The apparatus which I described in 1893, and had used for five years previously, was intended for the treatment of acute rheumatism or any condition where it was necessary to apply the process without removing the patient from his own bed. It consists essentially of a double-metal cover, which can be placed over the patient from the feet, where it is closed, to the shoulders. Between the two pieces of metal is a quarter-of-an-inch space which is filled with boiling water immediately before it is used. The patient lies upon a blanket, a hot moist blanket is placed over the front of the body, and then the cover is placed over the patient. For convenience, it is divided into two parts, one for the lower extremities and the other for the trunk. The result is that the patient is surrounded with warm moist air.

The effect of this appliance is to cause a rise of temperature of 2° to 3° F. Sweating usually commences in fifteen to twenty-five minutes according to the condition of the patient. When sweat appears on the forehead of the patient, it is the signal to finish the process. It does not increase the efficiency to continue it longer, and if sweating occurs too quickly it is a sign that the temperature used has been too high.

A large number of patients who require treatment have chronic valvular disease of the heart. The effect

is to stimulate the heart and improve the tone of the pulse. In the many thousands of treatments given during the last twenty-five years we have never had a case where the slightest untoward symptom has occured while the patient was on the thermal couch. Many patients over eighty years of age take daily treatment for a long period, and are not exhausted either at the time or subsequently, because fever, of short duration, is a most powerful physiological tonic. It is not unusual for patients taking the treatment for the first few times to have a feeling of oppression before the cutaneous excretion commences; but it is common to all baths which raise the body temperature, for the reason I have explained.

Immediately after the treatment the patient is usually given a spray or needle-bath, in order to restore the tone of the cutaneous blood-vessels. It happens rarely that patients may feel faint during this process, and it may be necessary, especially in cases of dilated heart, to substitute tepid sponging; but of course the great majority regard the needle-bath as the most enjoyable part of the process. A large number of patients go out of doors in all weathers soon after the treatment, but I have never yet traced a chill or a cold to this cause. The greater physiological activity of the skin has the effect of increasing resistance to atmospheric conditions.

Every patient has the night and morning temperature taken, and if the morning temperature begins to fall, it is an indication that the treatment is over-stimulating. Such cases are of rare occurrence, but I think all treatment of this kind should be accompanied by exact observations.

Usually patients feel distinctly better directly after

and during the course of treatment, long before the joint symptoms show signs of improvement; but it may happen that a patient feels a certain amount of malaise during the course or at some part of it. Usually in these cases there is a continued rise of temperature, produced by the process. It is so slight that the patient is not aware of it; but when it happens it greatly shortens the necessary duration of treatment.

A large proportion of patients have a subnormal temperature. If it rises to normal and continues at, or a little above, the normal line, it represents a true fever, and it will fall to the normal of the patient directly the 'fever' abates. We thus frequently produce attacks of 'rheumatic fever' which are only recognizable on the temperature chart.

Some patients who may appear in good health will complain of feeling exhausted after the bath, for no very obvious reason. In these patients there is almost always a spinal irritability, and the heat over-stimulates the nerve centres and produces exhaustion. A folded towel placed down the spine, by protecting it from heat, will prevent this trouble.

Whether we watch the effect of natural fever in eliminating lactic acid from the tissues, or the effect of pyretic treatment, we have to recognize that the process is a slow one. Most often in rheumatic fever the temperature falls and the joint symptoms disappear before all the acid is removed from the tissues, and unless we take steps to prolong the fever we are sure of subsequent trouble. In pyretic treatment we are not concerned with the improvement of the joints as an indication that we have attained our results. The real clinical indication is the reaction of the skin: until

it resumes the normal very slightly acid or neutral reaction, the patient is not cured.

On this point there is marked difference between the lactic-acid patient and the one with a lithic diathesis. The lactic-acid patient starts with a very acid reaction, and this will continue very slowly diminishing until the normal is attained. The lithic or 'gouty' patient will start with a neutral or slightly acid reaction, and this will become slowly more acid until a point is reached when the whole body may give a very acid reaction, and then it will gradually decrease in acidity until it is neutral or very slightly acid. When this point is reached we cannot say that the patient is free from lithates; it only means that we have set free all the available lactic acid, and this has, so far as it has been able, dissolved up these lithates.

If we continued the treatment under these conditions we should not make any further improvement. If the patient now takes active exercise, taking due care to maintain the skin in action, he will go on improving, and if at the end of six months he has another course of treatment, marked improvement will result. For this reason, while in rheumatism the best effects are obtained from a long course of treatment, for gout two shorter courses will generally yield better results, and may be regarded as necessary in most cases.

The value of pyretic treatment will never be fully recognized until it becomes employed by the practitioner in suitable cases. When this happens, we shall no longer have the patients with easily curable forms of rheumatism allowed to drift on until they become helpless cripples and a burden on society. This is not a theoretical statement, but the result of thirty years' daily experience. It is less easy to carry out

pyretic treatment than it is to write a prescription; but there are few households where all that is necessary for early treatment does not exist, and the only thing needful is the assistance of some person fitted to carry out the details of the treatment.

The *hot moist blanket pack* is an efficient form of pyretic treatment in many cases. The requirements are four blankets laid upon a bed, and a thinner blanket, well wrung out of hot water, placed on the top of these. The patient lies in the centre of the moist blanket, which is drawn round him, and then each of the dry blankets is in turn folded over the patient, tucked well in at the sides and round the shoulders. A hot-water bottle to the feet, and a down quilt to cover all, are of advantage. The only difficulty in this process is the moist hot blanket. It is best prepared by folding it, rolling it up like a rug used for travelling, and then placing it on end in a bucket. Boiling water is now poured through the centre to moisten it completely. It is then thrown into a large towel, and can best be deprived of all superfluous moisture, which is necessary, by two people twisting the towel at either end. It is this part of the process which may be difficult in some cases, when the necessary service cannot be obtained. The hot blanket pack can be improved upon, and a greater rise of temperature attained, by interposing a mackintosh sheet or an oil sheet between the first and second dry blankets. This prevents evaporation, and more efficiently prevents the radiation of heat from the body.

A simple method, well adapted for domestic use, without skilled help, is the 'foot-bath pack'. Four blankets are spread on a comfortable arm-chair, in such a way that they have six inches of their lower borders

on the ground. A foot-bath is half filled with hot water and placed in front of the chair. The patient, clad in flannel pyjamas or nightgown, sits on the chair and puts his feet into the hot water. The blankets are now folded *over the bath* and the patient, so as to prevent the escape of steam, and also so that no cold air is admitted. Care on this point is the essential factor in producing the result. In about ten minutes, more hot water is added to the foot-bath by opening the blankets just sufficiently to do so, and this may be repeated if required. In twenty-five to thirty minutes the temperature of the patient is usually raised about 2° F., and there is a good perspiration. The patient then gets into a blanket-bed, the feet only being *quickly* rather than *completely* dried. This is a very useful treatment when a patient in good health has been exposed to a chill, or for elderly or feeble invalids who require treatment with the minimum of fatigue. It is not efficient in cases where there has been prolonged functional inactivity of the skin, such as we find in the lithic diathesis, but it may be used to maintain functional activity when this has been secured by other means.

Although it is a simple method, its success, like all balneological methods, depends upon attention to detail and a clear understanding of the principles involved. The hot water raises the temperature of the blood as it circulates through the feet. The blankets, being wrapped closely around the patient, check the radiation of this heat. Therefore the higher the temperature of the water, and the more efficiently the packing is performed, the greater will be the rise of the body temperature.

In many cases of a chronic character the ordinary *vapour baths* give good results. The portable vapour

baths sold for this purpose have the disadvantage that the steam enters the cabinet too high above the floor, so that the feet remain cold. It is always advisable to have the patient's feet in a foot-bath, to which a little washing-soda may be added with advantage. In chronic rheumatism, if the smell is not objected to, a solution made by boiling sulphur with washing-soda, added to the foot-bath, is helpful.

One cannot attain the efficiency of more elaborate apparatus by these simple methods, but they are capable of doing an enormous amount of good if they are carried out persistently, if the practitioner is not deterred by some aggravation of the symptoms in certain cases, and, beyond all, if he does not expect the acid which may have taken years to accumulate to be removed in a week or two of treatment. I remember one case in my early experience. A physician who adopted my views with great enthusiasm, and whom I had supplied with special apparatus, wrote me to say that he had given a military man with chronic rheumatic gout twenty of my baths and he was still uncured; therefore, as he did not know what to do, he sent him to see me. I simply gave him twenty more baths, and he made such a good recovery that he was permitted to re-enter the Army during the Boer War, and subsequently became a distinguished general.

Another cause of failure is to mistake great improvement in the joint symptoms for a cure of the condition. The only reliable test is the reaction of the skin. I have already pointed this out, but it is of enormous importance. I am less concerned with the condition of a patient immediately after treatment than I am with his condition six months, or several years, later. It is the life-history of patients from which we learn

most in these cases. Thus a gentleman 82 years of age was very much crippled by rheumatic gout. After a month's treatment he was decidedly better, but still very infirm. Nine years later he came to me with pain in the muscles of the thighs, which I found to be due to his practice of walking over a very steep hill every morning as a constitutional. There was no trace of the original joint trouble, and at the age of 91 he was taking long walks every day which would have fatigued most men of middle age.

There is another form of pyretic treatment very valuable in certain cases: it simply consists in *active exercise* of any kind when the body is over-clothed with wool next the skin. Free action of the skin is produced and the temperature rises. The patient then removes the garments and sponges the body with hot water before resuming his ordinary garments. The treatment is only adapted to fairly robust patients and those of the lithic diathesis. It is not advisable for those of the lactic-acid diathesis, as, while it aids the elimination of lactic acid, it also increases its production. It is probably because muscular exercise increases the production of lactic acid that those who have accumulated too much are prevented from making more by the stiffening of their organs of locomotion. Nature is wonderfully protective in her methods, and uses lactic acid to prevent us from over-working our muscles even in health.

There is another method of pyretic treatment so simple that it hardly appears necessary to mention it, but which is of great value in overcoming the immediate effects of a chill. It is simply for the patient to lie on a blanket at night and have an extra amount of bed-clothes to cover him. This will often induce a reaction

accompanied by free action of the skin, and a great deal of future trouble may be averted.

The administration of vaccines may be regarded as a form of pyretic treatment when a decided 'reaction' is induced. Until proof can be given to the contrary, I am inclined to attribute any good results attained to the fever produced, rather than to the nature of the vaccine which caused the reaction.

Treatment by mineral baths is another form of pyretic treatment. The constituents of the mineral waters may vary; but most of them have a certain reputation in the treatment of gout and rheumatism, and all have a common effect in producing a certain amount of pyrexia during the bath.

It is usual at such spas to give a bath on alternate days, clinical experience showing that more frequent baths may exhaust the patient. Such clinical experience is entirely founded upon failure to administer baths on physiological principles. The temperature of the body may be raised, and lactic acid set free, without its excretion by the skin. This produces the feeling of malaise I have described. Or there may be free action of the skin and dilatation of the blood-vessels, and no means subsequently taken to ensure their contraction. In these circumstances the bath is relaxing. If baths are properly given there are very few patients, if any, who cannot take one daily with advantage. The importance of this is that the period during which patients remain for treatment is limited. Four weeks is regarded as a long visit, and if at the end of that time they have only taken a dozen baths, only a limited amount of lactic acid will have been eliminated, and the results are by no means as satisfactory as they might have been.

It is only when daily observations are taken of the reaction of the skin that we know anything of the results of baths, and when we do this we learn that acids which have been accumulating in the tissues for years are not removed by a dozen baths. In the lithic diathesis it frequently requires twelve baths to bring the patient's skin to its maximum of acidity, and if the baths are discontinued at this stage the patient is more liable to an attack of acute lithitis than before treatment.

Thirty years' daily observation of the effects of pyretic treatment of all kinds has greatly impressed me with the fact that cases of chronic gout, rheumatism, and arthritis, in which a hopeless prognosis has been given, are perfectly curable ; but it has also taught me that these results can only be attained by prolonged and systematic treatment adapted to the patients' physiological requirements, and as to this we can only be guided by exact observations. The mineral spas of Europe have done great good in many cases, but the results within their capacity have never been attained, because their routine methods have no scientific basis.

CHAPTER X.

GOUT AND THE LITHIC DIATHESIS.

We find lithates around the joints in persons who are in a state of good physical health, and such persons may never have an attack of gout. But, as I have pointed out, those who have a tendency to the formation of lithates have usually a subnormal temperature, and the skin may be more or less inactive. Although this is the case, it does not follow that complete inactivity of the skin is of necessity followed by the formation of lithates, or that a skin capable of performing its function is incompatible with their production.

When we shake hands with a person of the lithic diathesis we find the skin dry and smooth to the touch, and the muscles convey a sense of resiliency. When we shake hands with a patient having true 'rheumatoid arthritis' there is a marked loss of muscular resiliency. The contrast between the handshake of such patients and those of the lithic diathesis is so marked, that it is quite sufficient to distinguish between the two cases without any further examination. At a time when patients of the gouty and rheumatic class are commonly diagnosed as having 'rheumatoid arthritis', this ready means of distinction is very important.

We are so accustomed to be told that gout is due to an excess of acid in the blood that it may at first be difficult to appreciate the fact that one of the great factors in the formation of the lithates is the absence of free lactic acid in the lymph-space. This apparent

contradiction is due to the fact that the conditions causing the formation of the insoluble lithates are not the same as those which produce the attack of gout. The confusion of thought which exists is due to the fact that two separate physiological processes have always been considered as due to the same factors. Not only is this not the case, but also the formation of lithates in the tissues is not of necessity followed by the symptoms we recognize as gout. This point can be easily demonstrated under certain conditions.

A lady whose skin had been in a state of entire inactivity since infancy, and had become hard and dry from the accumulation of epithelial cells, and who had occasional attacks of dry eczema on the hands and face from the effects of exposure to air, was otherwise very robust and took a large amount of active exercise. An examination of the joints showed an entire absence of lithates. But if this patient was confined to her bed for a week from any cause, lithates appeared in all the large joints and gave audible evidence of their existence. Directly she resumed active exercise they at once disappeared, and she at no time had any symptoms of gout or swelling and pain in her joints.

It is obvious in this case that rest caused diminution in the flow of lymph to the tissues, and also of lactic acid, the natural solvents of the phosphate of lime in the dead cells, and the result was that the salt was partially oxidized and became less soluble. Directly exercise was resumed, sufficient lymph and lactic acid was supplied to dissolve those concretions which had not become stable, and they disappeared.

While the skin is more or less inactive in the lithic diathesis, the diminution of its excretory power is not a direct cause of the deposit of lithates, but because

the physiological inactivity of the skin is accompanied by diminution of lymph in the lymph-space. The result is that we have two classes of lithic patients so far as their joint conditions are concerned.

The patient whose skin may be normally active, but who never takes any exercise to excite its activity, will accumulate a large amount of lithates in the joints, and this accumulation may not of necessity be accompanied by any active joint trouble. By the process of oxidation the lactophosphate of lime becomes more and more insoluble, and the concretions take the form more of plaster-of-Paris than of chalk, because it is harder and drier. This is the feeling it conveys when the joint is put through its movements. The crepitations are frequently audible, although the examination may be painless. But when subacute inflammatory symptoms appear in one of these joints, the crepitations in this joint at once become softer, showing that the deposit is receiving a larger amount of moisture.

We have another class of lithic patient whose life may be sedentary but who regularly takes vigorous exercise. He may play golf or tennis. During such exercise his skin may act freely, but there is no tendency to do so in the intervals. We may find his temperature subnormal, and that he is rather sensitive to cold, but is otherwise quite robust. This man, during the sedentary periods of life, will form lithates in the tissues which are redissolved during active exercise. The result is that the act of exertion causes a large amount of soluble lactophosphate of lime to appear in the lymphspace; and even if free action of the skin allows some of it to be excreted, it does not continue long enough to get rid of the whole, and this has to find

its way by the lymphatics to the thoracic duct and hence to the kidneys, where it is excreted.

In these cases we do not, as a rule, find much deposit of lithates in the joints. If we do find them, it is usually in some tissue which at one time or another has been sprained through an accident and a certain degree of physiological inactivity has resulted, or we may find it in the great-toe-joint, or shoulder. If the lactophosphate of lime passes through the kidney in a soluble form, it gives no visible sign of its presence; but as I have explained in the chapter on uric acid, it may set up those chemical transformations which result in the excretion of lithates or uric acid in its insoluble form. This result is so common when a man of sedentary life takes active exercise that we rather expect it. It is only when it frequently occurs, and especially when not only uric acid is formed but also small concretions, that we regard it as a symptom of the 'gouty' diathesis. The continuance of this condition, even when the lactophosphate of lime in excess is passed in its soluble form, has a very injurious effect upon the kidney.

I am not in a position to state what exactly happens. The kidney may not show by ordinary tests any impairment of its functions; but when we test it for its capacity to excrete lactophosphate of lime, we find it is very deficient. This can easily be done by saturating a specimen of the urine with chloride of ammonium and then adding liquor ammoniæ fortis and noticing the amount of precipitate which falls when the urine has been allowed to stand for a little while.

It is not uncommon, in cases with no symptoms which would usually be attributed to gout, to find that the kidney is excreting no lactophosphate of lime, or only a very small quantity. In these cases the urine

is usually very acid, and there may be some irritation of the bladder; but, as I have previously stated, a symptom which sometimes calls attention to the failure is attacks of giddiness which may come on when the patient is at rest, and be worse when moving about. In the absence of other causes for this trouble it is always well to test the urine in the way I have suggested. Such cases point very clearly to the absolute necessity of restoring and maintaining the activity of the cutaneous excretion in patients of the lithic type. Although the kidneys may for many years perform the functions for which the skin is intended, the work thrown upon them will sooner or later impair their functional activity for this particular work, and as a natural result we have those cases where there is an accumulation of lactophosphate of lime in the blood and the lymph which we associate with uric acid, and all the symptoms which follow from it.

Between the two classes of lithic patients I have described, there are a great variety of gradations according to the life led by the patients and the conditions to which they are exposed.

Another characteristic symptom which we notice in patients of the lithic diathesis is the tendency for the hair to turn grey or white, prematurely. This symptom occurs in persons whose health and general constitution is perfectly sound. It is not due to the excretion of lactic acid, because examination of the scalp of such patients gives usually a neutral reaction, except during the act of sweating. I found in many cases that the reaction of the dry scalp tended to decolorize moistened litmus paper. This led me to suspect lactate of ammonia, and consequently I made a point of examining the cutaneous excretion of the

scalp, during the act of sweating, for ammonia. I have found it so frequently in such cases that I can say, with confidence, that the cause of the prematurely grey hair in such cases is the excretion of lactate of ammonia. The fact that patients with the lithic diathesis form a large amount of lactate of ammonia—and we find it with great frequency in the cutaneous excretion of the whole body—may partially explain their incapacity to dissolve the lithates in the tissues. It may not be, always, the absence of lactic acid, but the fact that when it is set free it becomes locked up as lactate of ammonia, and this would have no solvent action upon phosphate of lime.

The effect of cold, by limiting the amount of fluid in the lymph-space, is an important factor in causing the formation of lithates. Formerly gout was regarded as a disease almost peculiar to men; at the present time, in certain forms, it is more common amongst women, the reason being that the majority of women have a great objection to clothing themselves warmly. They appear to suffer less discomfort than men from the effects of cold, but the physiological effect is the same. The woman whose hands are always cold will at a later period show lithates around the joints, and especially if she does not take much active exercise. The condition most favourable to the formation of lithates is for a person to sit in a cold room until the surface of the body is chilled. The lack of lactic acid, owing to the inactivity of the muscles and the absence of fluid in the lymph-space, renders it impossible for the normal process of metabolism to be carried on.

There are two important facts to be remembered in respect of this. Rheumatism and gout are more prevalent in Great Britain than in any other country.

The houses in Great Britain are worse heated than any in the civilized world. It might be thought that Canadians, inured as they are to excessive cold in winter, would find the climate of this country much more acceptable. On the contrary, they complain of the great coldness of the British house, and suffer from it. This question is so important in connection with the prevention of gout and rheumatism that I do not consider it a digression to consider it.

In this country our domestic heating depends mostly upon coal fires and gas stoves. Both of these give radiant heat which warms the objects in the room but not the air which surrounds them. As a result we may burn our knees by sitting too close to the fire, while the surface of the back is chilled. Incidentally, the methods used to produce radiant heat allow 60 per cent of the heat to escape up the chimney in the flue-pipe.

On the Continent and in the Colonies domestic heating is attained more generally by the use of stoves and central heating. This gives convected heat which raises the temperature of the air of the room. If too high a temperature is produced and there is an absence of ventilation, this method is objectionable; but to use such methods, so as to secure a temperature of 60° to 65° in the room, and at the same time use the source of heat as a means of ventilation, is one of the most simple mechanical problems, and one which ensures the greatest economy of fuel. It is a curious fact that although economy of fuel is regarded by the Government as of great national importance, no effort has been made to instruct the public on these simple facts. That their desire to give instruction is very great is evidenced by the fact that recently thousands

of pounds were expended on advertisements directing the public not to poke the fire!

Having regard to the great importance of this subject, I made a series of experiments as to the actual amount of coal gas necessary to heat the largest dwelling-rooms when practically the whole of the heat was utilized, partly as radiant heat, and partly as convected heat. I found that a small six-burner gas stove using 10 to 12 cubic feet of gas per hour was not only ample, but more efficient than the ordinary gas-fire burning 30 feet of gas per hour. I accomplished this by making the stove independent of the flue pipe or chimney for its draught, so that it was no longer necessary for 60 per cent of the heat to escape up the chimney. But I found that the whole subject of domestic heating involved huge commercial interests, and reform is difficult. It is ridiculous to talk of fuel economy while the present conditions remain, nor is it possible to have properly-heated rooms when radiant heat alone is used.

Next to warm underclothing and properly warmed rooms as a means of preventing gout and rheumatism, I attach great importance to the body being well protected from loss of heat during the night. On the Continent the 'duvet' used to cover the body during sleep is exceedingly light, but possesses the power of checking the radiation of heat from the body much more efficiently than the blankets used in this country, and is sufficient to maintain the action of the skin during rest. I was puzzled at one time to account for the fact that in Germany, where the consumption of nitrogenous food is greater than in this country, and beer is drunk in large quantities, and the climate is less equable, gout was less prevalent than in this country. Apart from the fact that nitrogenous foods are not directly

concerned in the production of uric acid, and the rooms are better warmed, I believe that the gigantic down 'duvets' under which they sleep, and perspire, are the real solution of the question.

Another important factor in the formation of lithates is the lack of fluidity in the lymph and secretions. This may be due to the simple fact that the person does not drink enough fluid. In some of the most marked cases I have met with of this deficiency, the patient had been advised never to drink at meals, in order to improve digestion. The result was that no drink was taken either before or after the meal, and the total amount of fluid consumed in the day was much below the normal requirements of the body. In these cases both the blood and the lymph become thickened. This causes a difficulty in the blood finding its way through the small capillary blood-vessels, and such patients often present the appearance of anæmia, the complexion having a dark, pallid hue. In many cases I have found that these patients had been actually treated by iron tonics, with the result that headache and congestion were produced. If we take the specific gravity of the blood in these cases, it is frequently above 1060 and has a high degree of coagulability. In some cases the embarrassment of the heart has been so great that heart tonics have been used over a considerable period, without satisfactory results. I have cured a number of such cases, where the gravest prognosis has been given, with no other remedy than home-made lemonade in large doses.

I have long distrusted my own judgement in the diagnosis between anæmia and hæmoglobin excess, and never give an opinion without examination. This can be done in a few minutes by placing a drop of

the patient's blood in a mixture of chloroform and benzene, and then adding more of the one or other ingredient until the fluid is of the same specific gravity as the drop of blood. The specific gravity of the fluid, taken with an ordinary urinometer, will then give the specific gravity of the blood. Allowing for the variation of individual constitutions, we usually find a higher specific gravity in patients of the lithic diathesis than we do in those of the lactic-acid diathesis. In rheumatic patients it is usually very low, and this will explain the lack of oxidizing power in such cases.

Contrary to expectation, I have found many patients who live upon a fruit and vegetable diet largely, who have sometimes a pallid appearance, have a normal specific gravity of the blood with a full proportion of hæmoglobin. In addition to the importance of obtaining the necessary fluidity of the blood and lymph in lithic patients, the question of the amount of hæmoglobin is important in reference to diet. The patient with blood of a high specific gravity is better without too much meat, because it increases the hæmoglobin, which is already in excess.

The water consumed by lithic patients who live in districts where the water contains much lime is better boiled to get rid of the excess. There is no doubt that hard water, especially in some districts, increases the tendency to the production of lithates. On the other hand, water with an absence of lime favours the production of true rheumatoid arthritis. Therefore, for ordinary use, I do not think that distilled water is always advisable, even if the water is very hard. It usually results in too little fluid being taken, and is not always fresh unless prepared at home.

While the lithic patient has generally a good digestion,

he is more likely than others to suffer from those digestive disturbances due to the consistency of the bile being too thick, so that there is delay or irregularity in its passage through the gall-duct. Such patients may have periodic attacks of headache or bilious attacks, and in some cases may have severe pain in the gall-duct, or even the formation of gall-stones. Pain in the gall-duct is often referred to the region of the appendix, and many cases diagnosed as appendicitis are due to this cause. Such attacks are usually recurrent. In the intervals there may be a good deal of distention in the upper part of the abdomen. For the relief of this condition I have found a pill composed of ext. belladonnæ gr. $\frac{1}{8}$, podophylli resina gr. $\frac{1}{8}$, with or without aloin gr. $\frac{1}{8}$, act as specific if taken regularly twice or three times a week. But it is important in such cases that a good supply of fluid should form an essential part of the diet.

I have discussed in a previous chapter the chemical changes which lead to the formation of the insoluble lithates. We have now to consider the physiological conditions which cause the lithates to undergo chemical changes and produce the symptoms called gout.

We have to remember that every mass of lithates is in a state of cohesion with the living cell. We know that the cell-wall of the living cell can receive and discharge lactic acid without producing any symptoms, and that the process is constantly taking place under normal conditions.

Prior to the attack of gout there must be a large accumulation of lactic acid in the living cell-wall, and when this is suddenly liberated by heat or active exercise, and there is mechanical interference to its passage to the lymph-space by the presence of the lithic con-

cretion and the density of the surrounding tissue, some of it must combine with the alkaline lithates and cause a certain amount of softening and decomposition. It will be observed that the causes of the acute attack—heat and physical exercise—are just the reverse of those which lead to the production of the lithate itself. The symptoms produced by the addition of the acid to the lithate will vary with the amount added. We may have merely slight tenderness or pain coming on at night from the warmth of the bed, and disappearing in the morning, or we may have various degrees of pain up to the classical acute attack.

The primary effect of the addition of lactic acid is slightly to increase the bulk and soften the surface of the mass. This can be recognized if we manipulate the joint during the early symptoms of a subacute attack. The interference with the normal alkalinity of the lithate excites a certain amount of irritation of the living cells with which it is in contact, and this causes an increased flow of blood to the part, with effusion of lymph. The result is a solvent action on the periphery of the mass. There is increased heat in the part affected, with consequent further liberation of lactic acid, which excites more inflammatory action, and increased decomposition of the lithates.

The whole process can be observed in those cases where the mass of lithates affected are only covered by the skin. It will be noticed that the mass is the centre of an inflammatory area, where great effusion of lymph has taken place. The periphery of the mass of lithates becomes softer and finally has the consistency of milk, and absorption of the fluid is taking place and the mass becomes smaller. At first, the centre of the mass retains its normal chalklike

consistency; but, as the attack continues, the whole mass becomes softer. It looks now as if the mass would be dissolved and disappear; but I have never seen this happen. At this stage the attack suddenly ceases, the inflammation subsides, the effusion disappears, and what is left of the lithates resumes its normal consistency.

This appears to offer conclusive proof that the exciting cause of the attack cannot exist in the blood or the lymph. If it did so, the attack would continue until the whole of the lithate was removed, as in the cases when other peccant matters set up inflammation and the blood-cells are concerned in their removal. If the blood contained the cause which excites the attack, all lithates would undergo the same process of decomposition simultaneously; but, as I have pointed out, this does not occur. Masses of lithate in other joints are wholly unaffected by the process, no matter how long it may continue.

The whole evidence shows that the exciting cause of the chemical action is local, and that the quantity is limited, and becomes itself destroyed by the process. The inflammation caused by the attack of gout not only liberates lactic acid from the surrounding cell-walls, but also favours its elimination. This can be demonstrated by testing the reaction of the skin over a joint during the attack. It will be found to be intensely acid; but if we test the skin reaction some six inches away from the joint, it may only show a very slightly acid reaction. This proves not only that the attack is liberating acid from the tissues, but also how localized is the action of the skin in the elimination of the products which are formed in the tissues immediately beneath any part of it. It is for this reason that we

obtain different reactions of the skin in various parts of the body, even under normal conditions.

A case illustrating this point came recently under my observation. An elderly lady, of robust type, had an enormous collection of lithates round the finger-joints, so that some of them were deformed, but there was a singular absence of lithates in all the other joints of the body. This puzzled me very much, until she told me that many years ago her dress caught fire and she put up her hands to save her face from being burned; the result was that all the skin of the back of both hands and fingers was completely destroyed. The surface had healed in a remarkable manner, but of course all the sweat-ducts on the dorsal surfaces were destroyed. This fully explained the cause of her trouble and its strict localization.

Of course, in the more chronic forms of gout, where there is defective excretion of lactophosphate of lime by the kidney, the condition of the blood which results must exercise some influence upon the frequency and duration of the attack, as it does upon the general health.

In my experiments with lactophosphate of lime I found that to produce 'uric acid' we required two reagents, an *acid* and oxygen. Under normal conditions neither of these agents will produce any effect upon the oxidized lactophosphate of lime which forms the lithates; but when chemical transformation has commenced, the lithate irritates the living cells to which it is attached, and these cells are influenced by the condition of the lymph which surrounds them. This will explain the fact that a person whose joints may be crowded with lithates can eat red meat and take acid drink without the slightest symptom being pro-

duced, and will benefit, whilst the patient with lithates in a state of painless decomposition may experience almost immediate pain from some acid drink or increased act of oxidation.

But this condition does not occur unless the tissues are loaded with lactic acid to the saturation point and there is a physiological obstacle to its elimination. Directly such patients have a free action of the skin and elimination of the lactic acid, they can take red meats and acids with impunity. For this reason I have practically never occasion to diet patients when under my treatment. They make the best recoveries when they abandon strict dietary and take ordinary food.

Thus a gentleman, accustomed all his life to drink port wine at dinner, and who also took regular daily exercise, had a first attack of rheumatic gout in a number of joints at the age of eighty. He was treated for this at a spa, where abstinence from wine was part of his régime. His joints were unrelieved, and his health suffered seriously, and he came to me in a very weak state. I at once restored his port wine, gave him daily baths, and he made a complete recovery.

The port-wine-drinking habits of our ancestors as a cause of gout is a delusion firmly rooted in the public mind. It is forgotten that the labouring classes in those days drank large quantities of strong beer and did not develop gout ; yet beer is regarded as quite as potent a cause of gout as port wine. Gout was the aristocratic disease because it affected those who did not earn their bread by physical labour. It was not the port which caused gout, but the fact that our ancestors neither took baths nor kept their skins clean by the sweat of physical exertion. If we examine any large

mansion built a century ago, we shall find that no provision was made for a bath-room ; the necessity for it never occurred to the architect. The skin under such conditions would be so coated with dead cells that it could no longer become an organ of excretion, and so we had gout of a more active type than is known at the present day, when baths are regarded as necessary to civilization.

The question of alcohol in relation to gout has been discussed without any relation to the fact that alcohol may act as a direct stimulant to the tissues and the processes of metabolism, or as a cause of exhaustion, according to the doses which may be taken. We have to remember that a large proportion of the inhabitants of this country have a persistent subnormal temperature, and this is the direct cause of gouty and arthritic troubles. One of the most important physiological and therapeutic questions which concern the medical profession is to find some remedy which will restore the subnormal temperature. By doing so they would arrest the cause of a large amount of the disease and debility from which the British public suffer. I have been working at this subject during the last thirty years, and have failed to discover any agent except alcohol in *moderate* doses which can permanently raise the body temperature.

I am fully aware that writers have stated repeatedly that alcohol lowers the temperature, but I have found no experimental work to justify this statement. I am not concerned with the immediate effect of a dose of alcohol—this in any case must be very transient—but of the use of alcohol in small doses once or twice daily with the meals, over a period of some weeks. My experiments show that in a large proportion of cases

where the temperature is subnormal the effect of alcohol, used in this way, is to raise the temperature permanently. The results may not appear on the temperature chart for some days after the course has been commenced, and in every case it is gradual. This shows that it is not the direct stimulant action of alcohol which produces the result. It must have a definite food value in increasing the metabolic processes of the body.

I have tried to discover some other agent which will give the same result, but so far I have failed. I wish it were otherwise. I am quite certain that if prohibition were adopted in this country there would be a great increase in the number of patients of the arthritic class, and probably a century would elapse before the cause was discovered.

I think it quite possible that the excessive use of alcohol may cause or contribute to the production of arthritic disorders, because over-stimulation must produce exhaustion ; but I have had a singular absence of evidence on this point in my personal experience. On the other hand, I have been struck by the fact that the large majority of patients I have to treat, either take no alcohol at all or, if they do so, take it in very moderate quantities. I have, however, seen a good many cases where gout could be directly attributed to excess in food, and some of the worst offenders in this respect were men who boasted that they had been life-long abstainers and never smoked. I think smoking has one beneficial effect : those who indulge most do not consume too great a quantity of food.

When we are treating a patient with the lithic diathesis our object is to increase the production of lactic acid, set it free from the tissues, and ensures its

elimination. This we can do in robust men by active exercise under conditions which produce free action of the skin. But if we try the method on those who are not so robust, we shall cause fatigue, which is a factor to be avoided. Moderate exercise with pyretic treatment gives better results in these cases. In subacute cases of lithitis when some of the urates are being decomposed, active exercise is contra-indicated, as we do not need any surplus of lactic acid in the lymph. Pyretic treatment with copious, slightly alkaline drinks will relieve the irritation. Acids and acid fruits are to be avoided only during the duration of the attack. To deprive the lithic patient of fruit under ordinary conditions is very undesirable. In certain cases, sugar or red meats will, at this stage, by stimulating oxidation, increase the pain; but these, as I have mentioned, will only do so while the cells are overloaded with lactic acid. Directly we relieve this, they can be taken with advantage.

CHAPTER XI.

'ARTHRITIS.'

During recent years this word has been used to designate joint diseases of a widely different character, so that it has ceased to convey any information as to the pathological condition present, or afford any guide to prognosis or treatment. Thus, in Sarjous' *Analytical Cyclopædia of Practical Medicine* I find, under the heading, 'Chronic Rheumatism', chronic articular rheumatism or rheumatoid arthritis, rheumatic gout, rhumatisme chronique infectieux, polyarthritis deformans, and osteo-arthritis. This disease, which has received so many names, is stated to be 'due in all probability to the invasion of micro-organisms'. We have here a number of forms of joint disease, differing in their etiology, pathology, and treatment, classified as one disease and due to the same cause. It is not remarkable, under such conditions, that treatment fails and a grave prognosis is given in every case. The names in common use will do well enough to describe the cases met with in daily practice.

CHRONIC RHEUMATISM.

This is a disorder found in persons of a sound constitution and who are often robust. The patient may be of the lactic-acid diathesis, or have been in the habit of producing large quantities of lactic acid as a result of physical exertion. Thus the labourer in the fields is liable to suffer from chronic rheumatism, while

his master, the squire, under different physical conditions, is troubled with gout. Chronic rheumatism is much more likely to attack those whose skin excretion is unusually active. Thus a person resident in a hot climate, where excessive action of the skin is produced by the heat, will, on returning to this country, have a check given to the cutaneous excretion, and may as a result develop chronic rheumatism and become an absolute cripple for life because the cause is not understood.

The cause of chronic rheumatism is a chill which checks the excretion of the skin, no reaction taking place. It may be difficult to understand the failure of reaction in persons who are frequently robust, but clinical experience shows that the condition may continue for years without reaction occurring. When it does occur we have the symptoms of rheumatic fever, which is the natural method of cure. But in cases of chronic rheumatism, if we raise the temperature artificially we can seldom produce a continued pyrexia of an active character; the daily rise may be only two or three degrees above the patient's normal; in many cases no *continued* rise of temperature takes place, in spite of daily treatment. This indicates that the lactic acid in the cell-wall is in a state of chemical combination with the lactophosphate of lime, whereas under ordinary conditions a large part of the lactic acid is simply additive and easily liberated by a rise of temperature.

Chronic rheumatism has certain diagnostic symptoms which clearly distinguish it from other forms of chronic joint disease. If we examine the hand, we shall find that the fingers are partially flexed without any swelling of the joints. The knuckles will probably appear

swollen, but closer examination will show that this is due to wasting of the muscles and the tissues round the joints. The muscles waste in chronic rheumatism because the joints are disused, and also on account of the thickening of the cell-walls interfering with nutrition. The latter factor is important, and has never been pointed out. Although the muscles have wasted, they retain their resiliency and are firm on pressure. We find as the case goes on that both the elbows and knees become flexed and movement is considerably impaired. The arms become increasingly less capable of abduction and of being raised to the head, because of the stiffening of the shoulder-joint.

In some cases we may find symptoms which appear to justify the diagnosis of 'rheumatoid arthritis'. Some of the small joints of the fingers may be ankylosed, or have undergone such destructive changes that, instead of flexion, we have a slight backward displacement, so that a concavity appears over the dorsal surface of the joint. But this differs from rheumatoid arthritis in the fact that it is not the commencement of general destructive changes in the joints, but a purely local disturbance which is not likely to be repeated during the lifetime of the patient. We may also find in advanced cases a slight abduction of the hand from the wrist, due to muscular atrophy. This disappears as the patient recovers. In some cases there is synovitis of the knee-joints due to secondary symptoms or over-strain of the joint, but this does not alter the fact that swelling of the joints is not a *normal* symptom of chronic rheumatism.

Although the symptoms of this disorder are so characteristic, I have not seen a chronic case for many years which has not been diagnosed as rheumatoid

arthritis and received the treatment and prognosis of that disease.

Long clinical experience enables me to affirm that chronic rheumatism is a curable disease, even when it has existed for many years. Such cases involve much time and trouble, because it is necessary to treat not only the disease but the physiological and physical consequences. Chronic rheumatism is wholly due to physical causes, and can only be treated by physical agents which overcome the condition. It is in such cases that wrong diagnoses and the bacteriological theory have done an extraordinary amount of harm to humanity. Not only are vaccines useless in such cases, but they waste valuable time, during which the disease is making progress, and debilitate the patient when he needs all his powers of vital resistance.

The following case will illustrate chronic rheumatism in its most simple form, both as regards its cause and cure :—

A lady of middle age, and of a robust type, had lived most of her life in South America, in a very hot climate, where she had excessive action of the skin. She came to this country five years ago. She was chilled by the cold and damp, and began to develop stiffness and pain in her joints. This increased very gradually until her fingers had become flexed so that she could use them but little ; but, being a very energetic woman, she tried to use all her joints in spite of their stiffness. Her case was diagnosed as rheumatoid arthritis, and under advice she had all her teeth removed, in spite of the fact that they were perfectly sound. This, of course, did not help her, and after varied treatment she consulted me. The case was one of typical chronic rheumatism, and I ordered pyretic treatment, which I

considered would meet all the requirements of her case. I did not see her again for four weeks, and she told me she had only eight treatments owing to domestic difficulties ; but I was astonished at the extraordinary change in her physical condition : the stiffness had gone from her joints, and she was able to move her fingers freely, and this had happened in a much shorter time than I expected.

The nurse who gave the treatment told me that during the first six treatments the patient exfoliated an enormous quantity of epithelial scales. It is evident that, the action of the skin being checked, the dead cells had remained on the skin, forming an impermeable coating and an entire check to excretion. The treatment had set free the necessary lactic acid to remove this coating, which could not have been removed by hot baths or soaps. The patient was naturally healthy, and the restoration of her skin to a condition which enabled it to perform its natural functions enabled her to clear out the excess of lactic acid from the cell-walls. In this case the power of vital resistance of the patient was a great factor in the early success of the treatment.

Another case, which I have at present under treatment, well illustrates the unfortunate results of failure of diagnosis, combined with the bacteriological theory. It will be understood that I never blame the practitioner in these cases, because his only sources of reference do not provide him with the means of differentiation, and lead him to adopt a theory which is unsupported by either pathological or clinical evidence.

The patient was a healthy man who had not been away from business through illness for twenty years. He then had a slight attack of rheumatism, which was regarded as due to auto-infection and

promptly treated by vaccine. On the other hand, there was a distinct history of malaise for six months before the attack. The patient felt tired by his work, had a strong indisposition to take exercise, and used his motor to go to the station instead of walking. The symptoms were those I have described as the precursors of the rheumatic attacks. The following is the report sent me by his medical adviser :—

"At the end of 1913, and in January, 1914, Mr. X complained of pain and stiffness of toes. Subsequently was found with a large abscess round a tooth. In 1915 had abscess in throat. Erythema scarlatiniforme, with arthritis in fingers, toes, and ankle. Had vaccine administered. This did undoubted good, and was continued up to April, 1917. Ionization, sod. salicyl., and vaccine up to July, 1917. This last vaccine did not do much, and seemed to upset him. Since it was given up, a long course of ionization, massage, etc., has been tried, but his illness has progressed in spite of all."

The following is the patient's report of his illness :—

" First symptoms, pain in feet like pebbles in shoes; treated by Dr. A. with medicine.

" 1914. Pain down leg after sitting down. Dr. A. took me to Dr. B., who made vaccine from specimens (i.e., gums and fæces). After third inoculation, rash all over body, throat very septic, diagnosed rheumatic erythema.

" 1915. Continued inoculation with Dr. B.'s vaccine for about six months.

" 1916. Had a second lot of Dr. B.'s vaccine.

" 1917. January: Tonsils were taken out, as they were found very septic, and supposed to be the cause of my trouble. On fifth day after first tonsil was removed had a bad hæmorrhage from the throat and nearly went West. After some weeks had second tonsil taken out. I was a long time recovering from this, and was very weak for a long time.

" February : Had vaccine made from tonsils by Dr. C. May and June : Had a second lot of vaccine made by Dr. C. Developed pains in shoulders very acutely. July : gave up vaccine, and by end of August was very crippled and suffered much pain. Then went to Dr. D.,

who gave high-frequency for pain, which did some good; then ionization for some months.

"1918. Knees very bad; obliged to use crutches. *X*-ray treatment on knee, but no good result. Another vaccine by Dr. E. Local radiant heat and massage for couple of years.

"1919. Went to American osteopath for two months. Treatment very severe, knees terribly painful, nerves very bad from severity of treatment.

"1919. Went to Dr. F., who ordered me to walk. Ionization every day for eight weeks. Vaccine twice a week for sixteen months. Massage four days a week for eleven months.

"1920. July and August: Ionization by Dr. G. nine weeks, and massage four days a week. Thyroid gland to begin at 15 gr. per day and work up to 45 gr. When I had taken 30 gr. per day, heart got bad and was very depressed and nervous. When I left Dr. G., he had my teeth *x*-rayed and ordered them all out. This I did not have done. Returned to Dr. A., who treated me for my heart and nerves and recommended me to Bath."

I have reproduced the patient's written statement, only substituting letters for the names of the physicians attending. The condition of the patient on arrival was: The lower limbs were contracted to an angle of 45° and resisted all efforts at extension. Both elbows were also flexed. The shoulders permitted a very limited range of movement, while the ankles could be flexed and extended to their normal degree. The fingers were contracted and fixed, so that they could not be extended or fully flexed. Some of the small joints showed symptoms of degenerative changes, and there was some abduction of the whole hand. The joints were not enlarged or swollen, although the wasting of muscles gave the appearance of this condition existing. My diagnosis was pure 'chronic rheumatism'.

It will be noticed that, in spite of the fact that a source of auto-infection was discovered, vaccines used over a very prolonged period proved useless, and only

delayed other treatment. The practitioner may find sources of auto-infection in half a dozen patients a day, who have not, and do not, develop joint trouble. It is only when such trouble commences that the search is made for this source as a cause ; and if this investigation fails, then the bacteriologist can always fall back on the teeth, as few people have teeth beyond reproach !

The treatment adopted in this case and other similar cases of advanced chronic rheumatism is as follows :—

The additive lactic acid is first removed by daily pyretic treatment. This is continued for as many weeks as the excretion of the skin remains acid. Before all the acid is eliminated, pain during rest entirely ceases, and the tissues round the joints are softened, so that a greater range of movement becomes possible. This is increased partly by passive movement and also by directing the patient to move the joint, offering slight resistance to the movement, and, at the moment when the patient has completed all that is possible to him, giving such assistance as will increase the range of movement.

This requires great caution, because it is quite easy at this point to break down adhesions and cause the patient acute pain ; but if this is done the operator loses the confidence of the patient, and it will be difficult to make him use his fullest effort to move the joint again. The movement of assistance should be given very gently and slowly, and the patient should be directed to call out directly the pain becomes too great. If the operator immediately desists, the patient will feel confidence and will submit to a reasonable amount of pain to secure greater movement. It is at this stage that the success of treatment depends very much upon the efforts of the patient. It is for this reason

that the pessimistic prognosis is worse than the lactic acid, because its effects are frequently more difficult to remove.

I recently had a case of a lady who had been bed-ridden for five years with contracted joints as a result of chronic rheumatism. It had been so impressed upon her that she had 'rheumatoid arthritis' and was incurable, that even when I had straightened the lower limbs and restored the use of the knee-joints she would lie with them in the flexed position, which had become their most natural attitude, and refused to believe that she would ever walk again. It was not until I had put her on crutches, and taught her to rise from a chair without assistance, that I made her recognize the possibility of walking. From that moment she directed all her efforts to improving her powers of locomotion, and recovered rapidly.

These patients have nothing to do but improve their range of movement and muscular power, and they must be taught the best way of doing so.

As regards the shoulders, the ultimate goal is to raise the elbow as high as the top of the head, flex the arm over the head, and touch the ear on the opposite side, while the biceps muscle is firmly pressed upon the ear of the sound side. They can begin by using the hand to touch the mouth, the nose, the forehead, and the head, always striving towards the final result. Subsequently they should be taught to place the back of the hand of the affected side on the sacrum, and gradually raise it up the spine until the dorsal region is reached, keeping the hand in contact with the spine. When these two movements are accomplished the shoulder has resumed its functions.

For the elbow, the patient should be taught to make

efforts to touch the shoulder with the tips of the fingers, and then extend the arm vigorously, at the same time separating the fingers as widely as posisble. He should also make movements of pronation and supination of the hand.

The wrist is most conveniently exercised by placing the arm palm downwards on a small bedside table, so that the hand from the wrist projects beyond the edge of the table. The patient can fix the wrist himself, while he allows the hand to drop as far as possible and then raises it to the straight position. Subsequently he performs the same movement with a small book held in the hand to give resistance to the extension movement.

The small joints of the fingers should be exercised constantly by efforts to make a fist and then extend the fingers as widely as possible. A rubber ring pessary is often useful in cases where the flexor muscles require exercise.

The muscles of the hip-joint can be exercised by the operator holding his hand on a level with the top of the patient's toes, and then directing him to move the foot in a circle round it ; then to perform the same movement as if the hand was there.

For the knee- and ankle-joints, flexion and extension movements should be constantly performed so far as possible. Even if the range of movement is very limited, the effort is valuable, as it stimulates the nerve centres and the nerves and increases their blood-supply.

It is not advisable to allow more than six movements of any joint to be made at one time, especially in the early stages ; but these may be repeated five or six times a day, so that the patient who means to get well is kept fully employed without undue fatigue.

I very seldom think it desirable to break down joints under an anæsthetic. In some cases, where the joint has no movement owing to contraction and thickening of the structures round it, it can be done with advantage; or, if it is found impossible by means of extension splints to straighten a limb beyond a certain point owing to the strength of the adhesions, force may be required. But otherwise there is little advantage in obtaining a greater range of movement in a joint than the muscles can control. This is especially the case with the knee-joint. A patient may be able to walk with a slightly flexed joint, but be wholly unable to do so if the joint is straightened before the muscles are able to give the necessary support. In many cases of chronic rheumatism, even when the functions of the knee have been restored, the muscles may be too weak to permit the patient to stand, and it is necessary to use a splint to support the knee when the patient begins to walk.

This happened with the patient whose case I have described. After all the joints had been restored by the methods mentioned, the patient was able to walk on crutches; but owing to the extreme wasting of the muscles it was necessary to support the knees with splints. It may be many weeks before the powers of locomotion are restored. I always dismiss the patients when they have reached this stage.

There is a type of chronic rheumatism which differs from the pure form I have described because it is attended by great debility. It is usually accompanied by a moist acid condition of the skin which gives no relief to the symptoms. These cases usually occur as secondary to rheumatic fever, and convey the idea that while in ordinary chronic rheumatism the lactic acid

is locked up in the cell-walls in an additive form, these have a large amount of free lactic acid which excites excretion without the full functions of the skin being exercised. Under pyretic treatment the sweat is intensely acid, and, as this free acid is removed, the tendency to passive excretion ceases. In these cases pyretic treatment frequently causes a temporary continued rise of temperature, which is a mild attack of rheumatic fever, and in such cases the period of treatment is shortened.

The symptoms in these cases very closely resemble rheumatoid arthritis, because of the soft state of the skin and muscles.

In one recent case I hesitated to give a favourable prognosis, as the symptoms were so severe and the constitutional disturbance so great. The patient was a boy, 12 years of age, with a history of rheumatic fever. His condition came on while in a hospital, and it was difficult to find fault with the diagnosis of rheumatoid arthritis, because the deformity of the hands was so great. There was abduction of the hand, with backward displacement of several finger-joints. The knees were in a condition of extreme flexion, and both elbows and shoulders had very limited movements. But there was an absence of the symptoms which I regard as essential to the condition to which I think the term rheumatoid arthritis should be restricted and which I shall presently describe.

This boy had pyretic treatment on six days a week for nine weeks. During the whole treatment his skin reaction remained persistently very acid, only showing a diminution during the last week. During this time he gained nearly a stone in weight. His general health became good, and gradually the joints were brought

to the straight position without any forcible breaking down of the joints. The methods used were those I have described. In this case the boy was only able to walk on crutches by the aid of splints to his knees, when dismissed from the hospital. When once this stage is reached complete recovery is only a matter of time and exercise.

RHEUMATOID ARTHRITIS.

This name can be properly applied to a disease which is readily distinguishable from chronic rheumatism. If we start with the joint symptoms, it will be found that there is always effusion of lymph, not only extra-articular, but also into the tissues round the joint. This gives the peculiar fusiform appearance to the finger-joints which is frequently, but not by any means invariably, found. This condition does not occur in chronic rheumatism. In both diseases there is wasting of muscle, but while that of rheumatoid arthritis precedes the joint trouble, that of chronic rheumatism is secondary to it, and partly consequent upon it.

But the most marked diagnostic sign is the condition of the muscle itself. In rheumatoid arthritis it is soft, in chronic rheumatism it is firm and resilient. So marked is this symptom, and so generally are the muscles affected, that even in a case where the ankle-joint is alone affected, the diagnosis may be made by shaking hands with the patient. The hand of the patient has a soft jelly-like feel, and the bones are distinguishable because there is no resiliency in the muscles. In a large number of cases diagnosed as rheumatoid arthritis it is only necessary to shake hands with the patient, without any examination of the joints affected, to know that the diagnosis is wrong.

In rheumatoid arthritis we are dealing with a constitutional disease, and the joint symptoms are only a symptom of the condition. The patient with chronic rheumatism may be a person of sound constitution and robust health; the rheumatoid-arthritis patient is always debilitated.

The only disorders with which rheumatoid arthritis, in its early stages, can be mistaken for, is subacute articular rheumatism or chronic rheumatism secondary to rheumatic fever. Under certain conditions the latter cases may drift into those of true arthritis. Given an anæmic girl of the lactic-acid diathesis, and place her under conditions of great nervous and physical strain, or subject her to the shock of operations, destructive changes may take place in the joints and the condition of true arthritis may develop.

In subacute articular rheumatism we may have great swelling and pain in the joints, but no destructive change takes place. In rheumatoid arthritis the erosion of the synovial membrane commences at an early stage, and the whole tissues of the joint gradually become destroyed. The only product existing in the body which can destroy the tissues is lactic acid, and the patient with rheumatoid arthritis has a very acid cutaneous excretion. But this is also true of the patient with subacute rheumatism.

We know that if we place some meat or a small fish in a strong solution of lactic acid, the tissues become friable owing to the complete solution of the phosphate of lime. The lactic acid in the joint of the patient with rheumatoid arthritis has not that greater intensity which would explain the destruction of tissue. We know, however, that when lactic acid invades the cell-wall it is the phosphate of lime which neutralizes it

and holds it, so that it cannot invade the protoplasm of the cell. We know that in the healthy cell the invasion of an extra quantity of lactic acid has the the effect of causing stiffness of the tissues, with greater rigidity, such as we find in chronic rheumatism. But this is due to its combination with phosphate of lime in the cell-wall.

If we suppose that there is a great diminution in the normal amount of phosphate of lime in the cell-wall, it would account for the want of tone and resiliency in the muscle, and it would also account for the fact that the tissues of the joint when invaded by lactic acid are unable to neutralize it or prevent its action upon the protoplasm of the cells. This being so, we shall not require a higher degree of acidity than that which exists in ordinary rheumatism to produce the destructive changes in the joints. These facts led me to take the view that the constitutional condition which causes rheumatoid arthritis is one in which the power to assimilate the lime salts is deficient, or the supply of lime is insufficient, and that it is when this condition is coupled with the lactic-acid diathesis that we have the joint conditions peculiar to the disease.

Just as I found that people living in districts where the drinking water was very hard and contained carbonate of lime had a greater tendency to lithic deposits than others, so I have found that in certain districts where the water is soft and free from lime true rheumatoid arthritis occurs more frequently. My observations on this point have not been sufficient to enable me to speak positively, but I think it is one well worthy of further inquiry, and also as to whether there are not other sources of deficiency of lime in the dietary.

The importance of providing sufficient mineral

matter to feed the cell-wall is evident, and there is an agricultural problem which points to the same fact. It is found that if the soil is made rich in nitrogenous matter by fertilizers, it is possible to grow corn with large heads and heavy with grain ; but when this is done the stalk does not support the head, which falls to the ground, and therefore the result is not satisfactory. Now straw contains a large amount of lime in the cell-walls, and it is evident that a crop of corn makes a great drain upon the available lime in the soil.

On theoretical grounds I should suggest that the difficulty would be surmounted if, instead of relying wholly upon nitrogenous fertilizers, lime was regularly added to the soil where corn is grown. I have no knowledge of agricultural chemistry, but I think that both in medicine and agriculture the importance of lime as a necessary *food* should be recognized.

In 'rheumatoid arthritis', and in all cases where I have found great loss of resiliency in the muscles, I have found a solution of the phosphate of lime in phosphoric acid give remarkable results: so much so that I think it is the absence of lime from the diet, rather than the loss of power to assimilate it, which is the cause of the conditions found in this disease. The rapid pulse and the relaxed condition of the blood-vessels also become more normal when this remedy is given. I found that a more soluble phosphate of lime could be produced by the addition of a small quantity of the perchloride of iron. The following is the formula I employ :—

℞ Calc. Phos. gr. cxcij	Acid. Phos. Conc. ℥ xivss
Liq. Ferri Perchlor. Dil. ℥ij	Aq. ad ℥iv

Of this, ten drops are given in water thrice daily before meals.

It will be understood that I use this preparation in cases of true rheumatoid arthritis, because it is indicated by the symptoms present. It would do more harm than good if prescribed for cases which have received the name of rheumatoid arthritis but have none of the symptoms which I have described as characteristic of the disease.

It is of very great importance to remove the excess of lactic acid from the tissues by pyretic treatment. As this involves raising the body temperature and causing free sweating, some physicians have thought that it would have a debilitating effect upon a patient already weak, and whose heart is rapid. As a matter of fact, when the treatment is properly conducted, the patients improve in weight, and the irritable heart due to the excess of free acid becomes rapidly improved.

The prognosis of any case of rheumatoid arthritis depends upon the degree of destructive changes which have already taken place in the joints. Further destructive changes are arrested when efficient treatment is given ; the difficulty is to repair the damage already done.

Formerly I regarded it as impossible to obtain renewal of the tissues and cartilages of the joint when once destroyed ; but I have seen so many cases where repair has taken place and a useful joint has resulted, that I think it well worth while to give time and trouble to securing this end ; this is most difficult, but also most necessary, in the case of the knee-joint.

There is a physical point of importance in these cases. Under all circumstances where there is heat in a joint absolute rest is indicated ; but in these cases where there is slight heat—and it is seldom very great—it is necessary to give some passive movements every day,

otherwise there is a danger of ankylosis. But in a joint such as the knee, the weight of the body should not be allowed to be put upon the joint, and it is often necessary to use a splint which prevents eversion of the foot. In such cases, a continuous compress (moist), a proper splint, and daily passive movements, give slow but satisfactory results.

In many cases, if the joint is flexed to its fullest extent, fluid will be found ; and this must be removed by a blister as a preliminary to all other treatment. Ionization is a very inefficient remedy for this condition. In cases where there is neither heat nor fluid in the joint, rest of the joint so far as the weight of the body is concerned is imperative in the early stage, but passive movements must be given daily. A splint is only indicated if there is a tendency to deformity. As the patient improves, resisted movements may be given with advantage.

In many advanced cases of rheumatoid arthritis that come under my notice, I find that the actual progress of the disease has been for some time arrested. The joints which retain a measure of mobility are those which the patient has been obliged to use. It is for this reason that from the first indication of the disease the patient should be encouraged to put every joint in the body through its full movements daily. Nothing favours ankylosis of the joints in such cases more than the absence of their normal physiological activity. This does not mean that the patient should be taught to walk on an inflamed or impaired joint, or strain the parts which enter into their mechanism. Exercise must be slow and gentle, and is most valuable when it is the result of the patient's personal effort.

I have mentioned the debilitated form of chronic

rheumatism which sometimes follows rheumatic fever. In these cases the solution of phosphate of lime is a very valuable remedy, as it is in others with no arthritic tendency but with a debilitated state of the tissues and loss of resiliency in the muscles.

It is intelligible that when the cell-wall is so deficient in lime that it cannot resist the normal acids of the body, it may also lack resistance to micro-organisms. Therefore, if a patient exhibiting the symptoms of rheumatoid arthritis has any condition causing auto-infection, such as pyorrhœa or any other discharge, the bacilli may add to the mischief in the joints and make the treatment less efficient. Hence in these cases it is not unreasonable to look for such possible causes of infection and remove them if we can. But if the view is taken that these conditions are the *cause* of the disease and their removal is a sufficient treatment, the results will always be unsatisfactory, and grave harm will be done by the delay of treatment which in these cases is urgently required. In true cases of rheumatoid arthritis the destructive changes in the joints take place very rapidly, and the removal of the excess of acid and the proper nutrition of the cell-walls demands the first attention. It does not follow that even if a source of infection is discovered it is of necessity a cause of injury to the joints. Some means should be taken to ascertain this point before operative measures or vaccines are prescribed. I will explain the method I adopt in the course of this chapter.

In true chronic rheumatism I have never seen the course of the disorder influenced by the existence, or removal, of sources of infection, or by vaccines; I have only seen great harm done by the delay of necessary treatment. I believe in these cases the cell-wall is

proof against infection by bacilli. But in the debility and arthritic conditions following rheumatic fever, and in true rheumatoid arthritis, I think the possible influence of bacilli is a factor which may have to be dealt with.

Cases of subacute rheumatism in anæmic girls make rapid improvement when pyretic treatment is given; but if diagnosed as rheumatoid arthritis and treated with vaccines the results may be disastrous.

I was consulted about one case, a rather delicate girl of 18, who had an attack of subacute rheumatism. Her parents regarded her as too weak to undergo pyretic treatment, and sent her to London. Her case was diagnosed as rheumatoid arthritis, and her tonsils were removed and a course of vaccine given. No improvement took place. It was then decided that the remains of the tonsils must be enucleated. This was done, and more vaccine was given. As she did not improve, her uterus was curetted! When I last heard of her she was a physical wreck, confined to her bed, and her condition was attributed to rheumatoid arthritis!

CHRONIC LITHITIS OR GOUT.

The symptoms presented by a case of chronic lithitis or gout are so opposite to those met with in rheumatoid arthritis, that it is difficult to understand how any confusion can exist between the two disorders. The typical patient with the chronic and subacute forms of multi-articular gout has a sound constitution and good family history. The muscles are firm and there is no tendency to wasting of muscles, except any which may be due to loss of physical activity owing to a joint becoming incapable of use. The hair is frequently prematurely grey, the skin may be inactive, and the

temperature below normal; but this does not affect the muscular activity of the patient.

As I have previously pointed out, the presence of lithates, either intra- or extra-articular, is not of necessity accompanied by pain; but attention is usually called to the disorder either because one or more joints have become painful, or actual swelling of the small joints of the hand is noticed. This swelling is hard and nodular (except in the acute form), and is due to the deposit of lithates external to the joint and with or without some enlargement of the heads of the phalanges. There is an excess of phosphate of lime in the tissues instead of the insufficiency found in rheumatoid arthritis. To suggest that a case of this kind is due to the action of bacilli, and to order teeth to be extracted, and place the patient upon vaccine treatment, is an error which no practitioner should make. The symptoms we meet with are invariably due to the efforts of the organism to free itself from the lithates. These efforts may be inefficient, and put the patient to much pain and trouble; but the business of the physician is to assist the natural process of recovery, not to try and check it.

The acute attack gives us the indications for the line of treatment which may be used in the more chronic forms with success. In acute gout we have a rise of temperature, during which the patient may break into a sweat which is very acid over the joints affected. A process takes place by which there is effusion into the tissues around the lithate which tends to dissolve it, and at the same time there is liberation of lactic acid, as the examination over the affected joints shows.

Now we can reproduce these phenomena in chronic cases by pyretic treatment, and we can cause effusion

into the tissues round the lithate by continued electric vibration. For this purpose it is necessary to use a vibrator which rotates like a drill, except when it is placed in contact with the skin, and not one in which the vibration depends upon the movement of an excentric wheel. The effect of the former is to raise the temperature of the tissues to which it is applied, providing it is used at the required point for seven to ten minutes. The use of this appliance is contra-indicated in cases where there is any heat on the surface of the joint because, in such cases, it would set up too much effusion in the joint and produce an acute attack, which is undesirable. Like all treatments for joint cases, it requires steady perseverance to remove the lithates from the tissues. The immediate effect is often remarkable, because a joint stiffened and painful to move becomes readily movable and painless after a single vibration. This is due to the fluid effused into the tissues. As this is absorbed, the stiffness returns, but each time some solution of the lithates takes place, so that finally they are disposed of.

In the acute cases where this treatment would be contra-indicated, the application of a strong solution of salicylate of soda, well rubbed into the skin, followed immediately by the liniment of iodine or the tincture, applied as a paint, is very useful. The chemical action taking place in the skin produces results which would not be obtained by mixing the two solutions before they were applied.

I have described the conditions under which the lithates are formed in the tissues, and also the processes which take place when the lithates undergo decomposition, and the symptoms produced. I have already suggested that when we find lithates in or around a

joint causing no pain other than any which may be produced by their mechanical presence, we should describe it as a lithic joint and the patient as having the lithic diatheses. When there is pain owing to the lithates undergoing decomposition, we may speak of it as acute or subacute lithitis. I think the word 'gout', expressing as it does a wrong idea of the cause and nature of the disorder, is undesirable. I regret that I have been compelled to use it in the title of this work.

There are other symptoms connected with subacute lithitis which will be best described in the chapter on 'neuritis'.

RHEUMATIC GOUT.

This name, although it has gone out of use, does well enough to describe a form of chronic joint trouble frequently met with. In this disorder there is a good deal of thickening of the tissue immediately round the joints. This is solid, and not fluctuating as in rheumatoid arthritis. It covers a larger area, and is more irregular than the enlargement of the small joints of the hand which we find in chronic gout or patients with the lithic diathesis, who may never have any symptoms of gout in spite of the lithates round the joints.

Some writers have doubted the co-existence of rheumatism and gout. It does not appear probable that the two disorders would exist in the same person, because a deficiency of lactic acid leads to the deposit of lithates, while rheumatism is due to an excess of the acid. But clinically we meet with cases where minute lithates which have existed in the tissues surrounding a joint have become involved in an acute or subacute

attack of rheumatism, and as a result the swelling of the joints characteristic of rheumatic gout has been produced. For some reason which I do not clearly understand, this may happen at the end of an attack of acute rheumatism when all the typical rheumatic symptoms in the joint have subsided. More usually it occurs at an early stage. The following case is very instructive in this connection :—

A physician in the South of England wrote to me about a young lady who had a number of swollen joints. He took her to a great specialist, who pronounced the case one of rheumatoid arthritis, and gave the opinion that she would be crippled in every joint within two years. Soon after, sugar was found in the urine of the patient, and she was taken to another specialist, who took a very grave view and thought that she would not live two years. But the physician had been treating her with iodide of iron, and instead of growing worse she appeared to improve; he therefore asked me to see if anything further could be done for her. On shaking hands with the patient, a healthy-looking girl, she gave me a firm grip which at once dissipated the idea of rheumatoid arthritis. On triturating the tissues with the finger over the affected joints I could clearly make out a number of tiny particles like firm sand in the tissues. I formed the opinion that she was suffering from subacute rheumatism, and that the action of the lactic acid upon these minute lithates accounted for the appearance presented by the joints ; a subacute lithitis had been set up. I gave her pyretic treatment for one month, and at the end of this time she was quite well, both as regards the joints and the glycosuria. I suggested a further course of treatment in six months, but at the end of that time she remained

so well that it did not appear necessary. The formation of lithates and the existence of glycosuria both pointed to 'gout', but the joint symptoms were 'rheumatic'.

Rheumatic gout must be regarded as a very distinct disorder, and is an attack of rheumatism in a patient who has lithates already deposited in or around the joints. As I have shown, a person may have lithates in various tissues for many years and never have an attack of rheumatism or gout. The attack of gout is most likely to occur when there are concretions of lithates in a joint, and it occurs usually in one or two joints as a local affection due to the condition of the tissues immediately surrounding the concretion, so that other joints in which lithates exist are not attacked.

But when a patient with lithates in the joints is attacked by rheumatism, it is due to the circulation of free lactic acid, and therefore a large number of joints are attacked simultaneously, and in many of these joints the lithates may be so small and scattered in the tissues that it is difficult to detect their presence. The skin of the patients when so attacked has the acid reaction and the soft adhesive feeling of the rheumatic patient, while that of the gouty patient is very slightly acid in reaction and only gradually increases in acidity, and the skin is brought into action and lactic acid liberated from the tissues by treatment.

While during the period when the lithates are deposited in the joints the patient may have a deficiency of lactic acid, there is no physiological reason to prevent the same patient manufacturing an excess of lactic acid under changed conditions. In fact this does occur normally when the patient takes vigorous and unaccustomed exercise; but the usual result is simply to dissolve the lithates without the production of pain or

discomfort. It is only when the tissues have become saturated with lactic acid that a true rheumatic attack is produced, and then the presence of the lithates influences the attack and alters the appearance of the joints.

The treatment of rheumatic gout is the same as for rheumatism either in its acute or chronic form. When we have removed the excess of lactic acid from the tissues, the attack ceases, whether all the lithates have been removed or not.

There is another class of case closely allied to rheumatic gout in which, instead of a firm swelling of the joints, the condition is one where there may be little actual enlargement, but the tissues around the joint have a puffy appearance which gives the appearance of considerable swelling. These cases have some resemblance to a joint swollen from the effect of myxœdema. There is swelling without effusion of lymph, but the form is less regular.

The actual joint symptoms may be those of chronic rheumatism or rheumatic gout; but these cases have less tendency to clear up with the same treatment, although they derive benefit, the pain being relieved and the movements of the joints improved.

I formed the conclusion many years ago that these cases were complicated by the existence of micro-organisms in the tissues, probably what we now recognize as streptococci and staphylococci. I did not reach this conclusion as a result of bacteriological examination, because it was before the bacteriological theory of rheumatism was introduced, I formed the view from clinical experience.

To trace the history of such cases we have to go back to the early life of the patient. We can usually

obtain information of some skin disease or the sudden suppression of an eruption. We next may get a history of some ailments other than the joint trouble, which may only appear at a later stage in life. One of these ailments is a headache, recurring at regular intervals—it may be one day in the week or fortnight—and this condition may persist for many years. About the age of 45 or 50 the headaches may cease, and the patient may develop the form of joint disease I am now describing. But I have known these headaches continue to the age of 68 and grow worse. I cured one such patient in whom the attacks had existed since she was 9 years of age.

The appearance of joint symptoms is by no means an invariable result of this disorder. Such symptoms only occur in those who have the lithic or lactic-acid diathesis, and who would have developed joint symptoms in any case, but not in the same form.

I have never taken the view, nor do I now in spite of all that has been written, that any micro-organisms can *cause* joint disease ; but I am equally strongly convinced that they can alter the character, increase the severity, and prolong the duration. It is just as likely that the conditions I have mentioned will cause an intractable form of asthma, and that this may occur in patients with lactic-acid diathesis, who might be expected to develop joint troubles.

Since writing the last paragraph I have seen a lady who suffered from these recurrent headaches during the whole of her early life. In 1903 she was vaccinated during a small-pox scare. Inflammation occurred at the site of vaccination and spread to the whole arm, and for nine months she had a series of 'abscesses' and eruptions in all parts of the body. Her condition was

diagnosed as blood-poisoning ; but in spite of all her sufferings her health was better than it had been for years, and the headaches entirely ceased. I take the view that the bacilli which caused all the trouble were not introduced from without, but that the vaccination set up an effort of the organism to throw out the bacilli which had existed in the tissues since early life, and had caused the symptoms from which she suffered. I have ample grounds for this belief on the evidence of clinical experience. I mentioned that I cured a lady of these recurrent headaches at the age of 68. I did so by artificially producing eruptions over the abdomen and in various parts of the body, and repeating the treatment for many weeks. I have done the same in a very large number of cases, with invariable success.

My attention was first called to the connection of this condition with asthma by the following case :—

A lady, who was a chronic asthmatic, had some pain in the region of the hip, and rubbed in a well-known embrocation, as a remedy. The result was a large inflammatory area over the site of the application, closely resembling erysipelas. This spread down the whole limb and subsequently invaded the opposite side. She had a good deal of pain and suffering, which lasted for nearly three weeks. At the end of this time the skin was healed and the asthma had quite disappeared. She lived for many years after this, and the chronic asthma remained entirely cured.

This experience induced me to try the effect of an artificial eruption in other cases. I found that in cases suitable for this treatment—that is to say, those who had staphylococci in their tissues—a few days after rubbing the skin with a liniment composed of linimentum crotonis and linimentum saponis, equal parts,

a pustular eruption developed. In some cases the application only set up a dermatitis, and I did not persevere in those cases. But when the pustular eruption was produced, I maintained it by mild applications from time to time so long as it could be made to persist. Long-continued treatment was necessary to obtain permanent results. The following case has many instructive features :—

A boy at the age of 5 had measles ; it was followed by asthma, which continued until the time of admission to hospital when he had reached the age of 16. He was much stunted in growth, and weighed only 4 st. 7 lb. The attacks during the previous three years had greatly increased in severity, with continual cough between the attacks. During the attacks there was great dyspnœa, violent coughing with expectoration, and profuse perspiration. The liniment I have mentioned was applied to the whole of the front of the chest and abdomen, and produced a crop of pustules over the entire surface. This was maintained for eight weeks. During this time the temperature was artificially raised about 3° each day, and profuse sweating produced. The reaction of the cutaneous excretion was intensely acid, and remained so until the twenty-eighth application of the process ; it then diminished until the fortieth administration, when the boy had a slight cold and the reaction became excessively acid and remained so for six days ; it then fell to normal.

This case shows that the boy had a very marked lactic-acid diathesis, and also the large number of baths which are necessary to clear the tissues of the accumulated acid. As the boy was in a debilitated state on admission, those unaccustomed to the treatment would think that eight weeks of daily baths, each

causing a rise of temperature and profuse sweating, would have increased the debility. His condition at the end of this period was as follows. The attacks of asthma, which had been milder and less frequent during the early stages of treatment, had disappeared for a fortnight. The boy looked perfectly well and expressed himself as feeling well. His weight was 5 st. $6\frac{1}{2}$ lb., showing a gain of nearly one stone. The circumference of the chest had increased by four inches.

The introduction of cases of asthma into a chapter on arthritis may appear to be a digression; but, in the class of joint disease I am considering, the pathological conditions appear to be identical so far as the cause of the disease is concerned. When we meet with such cases—and they occur with some frequency—it is always well to test the reaction of the patient to stimulating liniments, and if there is decided irritability of the skin we can be sure of benefit resulting from their persistent application, whether over the affected joints or at some more convenient part of the body. It is in this particular class of case that vaccines may be expected to do good. They may accomplish good because they antidote the cause of irritation which maintains and increases the joint trouble and leads to its indefinite continuance; but no vaccine can remove the excess of lactic acid from the tissues, and therefore it can only effect a cure when it excites a reaction resulting in a continued rise of temperature, or, in other words, induces the condition which nature uses to eliminate the acid.

But personally, I have never felt justified in setting up a toxic fever when one can accomplish the same result by physiological methods, and at the same time improve the health of the patient, which a toxic fever

never does. The combination of mild doses of vaccine with pyretic treatment may prove an efficient method of dealing with such cases; but the method I used before the bacteriological theory was introduced has given such good results that perhaps I am prejudiced in its favour.

ARTHRITIS DEFORMANS.

There is another form of joint disease for which the name arthritis deformans appears very suitable. In all cases where destructive changes take place in the joint there may be a certain amount of deformity—flexion, abduction of the hand, or backward displacement of the finger-joints; but in this disease the deformity is the most marked and characteristic feature, and in pure cases there may be very little swelling of the joint, no tendency to the lactic acid diathesis, and an absence of lithates. As, however, this disorder may attack persons of either the lithic or lactic-acid diathesis, the condition is sometimes more complicated.

When we meet with this condition there is almost invariably the history of either some nervous shock or prolonged nervous strain combined with physical exhaustion. In pure cases, the destructive changes in the joints are not due to the erosion of lactic acid as in rheumatoid arthritis, but to an asthenic condition of the nerves supplying the joint. We are dealing in these cases with a direct disease of the central nervous system, and while the symptoms may simulate those of rheumatism and rheumatoid arthritis, the pathology of the disorder is wholly distinct. The only source of confusion arises from the fact that while in the true cases the asthenic condition of the articular nerves is of central origin, we may have cases of joint trouble

where an asthenic condition of certain nerves is purely local, and only affects the area of distribution of the particular nerves affected.

To illustrate the pathology of such cases, I may mention the case of an officer of Horse Artillery. He had nothing the matter with his joints, but he had on the outside of the left knee-joint a small area of skin which had shrivelled and become entirely numb. The skin had a blanched appearance, and the surface resembled the goose skin condition which follows the application of intense cold to the skin. The trouble corresponded exactly with the area of distribution of the external cutaneous nerve. I found this nerve practically paralyzed. The cause was obvious: when riding over difficult country, in command of a mountain battery, the hilt of his sword was continually percussing this nerve and had induced the condition. This case shows that injury to a nerve-trunk may set up trophic changes in the area of its supply. Thus atrophic disease of the hip-joint is due to definite injury or pressure upon a branch of the obturator nerve in the pelvis, as I shall presently mention.

Another case well illustrates the fact that a local arthritis deformans can be produced by injury to the nerves. A lady, while travelling abroad, injured her wrist; the local surgeon consulted said that it was fractured, and kept the wrist and hand in a splint for a considerable time. When it was removed the patient's hand presented a complete picture of arthritis deformans; the joints were not merely flexed and stiffened, but completely ankylosed. All the other joints of the body were perfectly healthy, and the lady, although elderly, was quite robust. The fact that destructive changes took place in the joint points to

an injury to their nerve-supply. Simple fixation of the joints for a few weeks could not have produced ankylosis and such marked deformity.

In all cases where I find arthritis deformans as a general condition, I suspect the history of some shock to the nervous system or of a period of over-physical exertion combined with mental anxiety, and almost always find it. Thus a woman of middle age, and of apparently healthy constitution, had joints which were exceedingly distorted. She was the wife of a captain in the mercantile marine, and was accustomed to voyage with her husband. On one occasion during a terrific storm, when the position of the ship appeared hopeless, it was discovered to be on fire. For forty-eight hours she did not know whether she would be drowned or burnt to death. Immediately afterwards the joint trouble commenced and the joints became distorted. There was no history of rheumatism, nor evidence of lithates.

The fact that nervous shock can produce a serious disease of the joints, and may in a modified form greatly injure their nutrition, should be taken into account when delicate women are ordered to have all their teeth removed in the hope that it may favourably influence some joint trouble. We have no evidence to show that bacilli can initiate joint disease; but we have abundant evidence that nervous shock can do so.

I do not know of any treatment which will cure true arthritis deformans. In ordinary cases, where a local or general asthenic conditions gives rise to deformity, much can be done by appropriate splints to prevent it. It must be remembered that the disease is not progressive, but comes to a natural termination after a time. A large number of the cases I see are no longer

suffering from the disease, but from its after-effects, which are purely physical and may be improved by physical methods. Many of these patients have received vaccine treatment long after the original disease had ceased.

We may call arthritis deformans incurable because we cannot overcome the physical ravages it leaves; but it is really a disorder which comes to a natural termination, and is not so progressive as ordinary chronic rheumatism.

GONORRHŒAL RHEUMATISM.

There is one form of joint disease in which the influence of bacilli in increasing the severity of the symptoms, altering the character of the inflammation, and prolonging its duration, is unquestionable. This is gonorrheal rheumatism. The effect of the gonococcus is to produce considerable effusion round the joints, and, in cases where the streptococcus is also present, the tissues round the joints become very puffy, so as to make the swelling appear greater. But the gonococcus cannot be regarded as the cause of the attack, because not one patient in a hundred whose tissues are invaded by it develops joint symptoms. Neither does it usually happen if the patient has a well-marked lactic-acid diathesis. But given this diatheses, and an attack of gonorrhœa, and then a chill to check the excretion of lactic acid, we get gonorrhœal rheumatism.

It is for this reason that while two of the factors are very common, viz., the lactic-acid diathesis, and the invasion of the gonococcus, gonorrhœal rheumatism is fortunately a comparatively rare joint disease, because it requires the intervention of a chill or some cause which checks excretion. This indicates that all persons

with gonorrhœa should be warned of the absolute necessity of avoiding a chill. Thus, a soldier with gonorrhœa should not be exposed to damp conditions if it can be avoided. If in such cases the excretion of lactic acid is interfered with, and an acute or subacute rheumatism follows, the presence of the gonococcus has a very disastrous effect upon the inflammation of the joints.

If such cases are treated in the early stages by methods which liberate the lactic acid from the tissues, they give very little trouble; but if the action of the gonococci, and the presence of the acid in the tissues, are allowed to continue, destructive changes may take place in the joints and permanent injury result.

In one case, where I saw the patient within fourteen days of the commencement of the attack, the knees were chiefly affected, and the bursa above the knee-joint on both knees was enormously distended with fluid. In this case I gave pyretic treatment to remove the lactic acid, and at the same time adopted a treatment which may be regarded as a modified vaccine method. With a large syringe I drew off the fluid from the enlarged bursa, removing all that I could bring away. I then, without removing the needle, after sterilizing the syringe, injected a 1–2000 solution of biniodide of mercury (dissolved with iodide of potassium) into the sac. My theory was that this would destroy the gonococci present in the sac, and these would act as an autogenous vaccine. Whether my theory is correct or not, the results were very remarkable. At the end of three weeks the patient's knees were practically normal, and he was able to resume his occupation and had no further trouble. The effusion round the affected joints makes the treatment quite

practical in all cases, and, when seen before destructive changes have occurred, the results are good.

I am rather afraid of gonococcal vaccine, for I had one case of mild gonorrhœal rheumatism in a young undergraduate where it had been used by a physician to whom he was taken, and the result was disastrous. Not only was there no improvement in the joint symptoms, but he developed rupia, which covered the whole body, including the face. The boy was in a terrible condition. If vaccines are used in these cases it appears advisable that these should be autogenous, as it is difficult to be sure of the purity of a vaccine taken from another individual. I think the method that I have suggested will be found more practical as well as safer; but I do not think it would be of the same value unless steps were taken at the same time to remove the cause of the rheumatic symptoms, i.e., the acid in the tissues.

ARTHRITIS OF THE HIP-JOINT.

The chronic diseases which affect the hip-joint differ very materially from those we find in the other joints. True rheumatoid arthritis may attack the hip-joint, and when it does so it is usually one of the last joints attacked by the disease, and therefore offers no difficulty as regards diagnosis. The hip-joint may also be the site of lithic deposits, but less commonly than the joints which are more exposed. The forms of chronic hip disease that come most frequently under my notice are usually not associated with disorder of other joints, and may occur in cases where no other joint trouble exists. The term 'arthritis of the hip' is used to describe both these diseases; but they are very distinct in their pathology and symptoms.

The first I call *atrophic disease of the hip*, because it

is due to atrophic changes in the head of the femur, which gradually becomes absorbed. It is usually regarded as a disease of advanced age. I think, however, the first symptoms commence soon after middle life; but it is so slow in development, and so obscure in its physical signs, that it is rarely discovered until late in life. There is at first very slight pain on commencing to walk; this disappears very soon, and the patient is able to take a long walk. After a time any extra exertion is followed by pains in the thigh, which may come on during the night. Some time elapses before this symptom makes it necessary to curtail the daily walk.

If at this stage, when perhaps the disorder has existed for two years, a physical examination is made, the results are wholly negative. The patient has perfect freedom of movement of the joint, and there is no crepitation or pain elicited by any movement. After a time, a very characteristic symptom develops. The patient on rising from a chair, makes an almost imperceptible pause before he commences to walk. As the case goes on, crepitation can generally be elicited by flexing the extended limb on the body, and then offering strong resistance as the patient extends it. During this time the pain, chiefly referred to the thigh, increases after slight exertion. No points of tenderness will be found in the lumbar plexus or the nerves of the thigh. There is gradual shortening of the affected leg, with some eversion of the foot; but during the whole time the free movement of the hip is maintained; it never becomes ankylosed or even stiff.

I believe that the disease is due to pressure on the obturator nerve in the pelvis, causing an asthenic condition of the branch which supplies the head of the

femur. When it occurs on the left side I consider it is frequently due to chronic constipation. There are numbers of people who, in spite of a daily evacuation, have always a loaded lower bowel. In one case, where it occurred on the right side in a lady, there was the history of an abscess in the pelvis as a result of appendicitis.

In these cases rest of the hip-joint is essential. I have devised some mechanical appliances; but on the whole I think if the patient can be induced to use crutches, and so remove the weight from the joint, and use a Merlin chair for indoor use, the requirements are sufficiently met. But patients will rarely submit to the treatment until the disease has reached an advanced stage. In one case, where I recommended it and persuaded the patient to give up having useless courses of treatment, I did not see her again for some years. Then a physician, with whom I was lunching in London, asked me to see a patient of his. It was this lady, who had been trying one treatment after another since she had seen me, and was steadily getting worse. I have had a few cases where rest, persevered with for two years, has arrested the disease, so that no further trouble was experienced. In all cases rest is necessary to relieve pain.

There is another form of arthritis which I call *hypertrophic disease of the hip-joint*. This is the more common of the two. It usually, but not invariably, comes on after an injury, and consists of a general hypertrophy of the bones forming the hip-joint. It is attended by stiffness in the joint from the first, and the range of movement is gradually diminished. While rest may relieve pain, it is less necessary, because ankylosis of

the hip is the natural cure, and, when this happens, pain ceases.

One patient, a lady, had this disease in both hips, and the pain was very acute. I devised a splint which, by keeping the joints at rest, gave relief. Now both hips are ankylosed, and there is entire absence of pain. She can walk fairly well, and can even manage to mount stairs by twisting the body as she mounts them.

CHRONIC SYNOVITIS OF THE KNEE-JOINT.

A number of patients come under my observation with pain in the knee-joint which is aggravated by walking and descending a hill. In many of these cases there is no marked swelling of the joint; but if the knee is flexed upon itself and palpated, there will be found a marked tension or even bulging on either side of the ligamentum patellæ, just below the condyles of the femur. This indicates the presence of fluid in the joint, which has escaped observation because it gives no evidence of its presence when the joint is examined in the ordinary manner. These cases have usually been diagnosed as arthritis, and a grave prognosis given. Many of them have been treated by ionization without result, also by local hot-air baths and the local application of iodine.

The most efficient treatment is the application of two blisters over the sites of the swelling. This removes the fluid and practically cures the condition in forty-eight hours; but it must be remembered that when the fluid has been a source of irritation over a long period the tissues of the joint remain irritable, and a period of rest should follow the blister until this has wholly subsided. In some cases I have seen serious mischief to the structures of the joint from the result

of a little fluid left after an attack of acute synovitis from any cause. While inflammation exists the fluid is absorbed, but in the absence of inflammation the synovial membrane appears to be incapable of absorbing the fluid, and it may remain in the joint to be a source of irritation for years, unless there is a return of the acute symptoms.

Some patients with persistent pain in the knee-joint, worse on walking, and who have no fluid in the joint, are suffering from a subacute inflammatory condition which has not been detected, with the result that all treatments have failed, because the patient has been walking with an inflamed joint during the whole of the treatment, so that a mechanical obstacle to cure existed. This can always be avoided if the surface temperature of the joint is taken with the palm of the hand, and compared with the tissues above and below, as a routine method.

My own routine method of examining a knee-joint is first to take the surface temperature, next fully flex and extend the joint, and finish the movement in the position of extreme flexion while I examine the tension. The examination occupies less than a minute, but it gives information about lithates, arthritic changes, inflammation, or presence of fluid, and enables a diagnosis to be made with a certainty that would be lacking if we had only an *x*-ray photograph to guide us.

These subacute inflammatory conditions of the knee-joint, especially when they have existed for a long period, do not yield very readily even when absolute rest is given to the joint. I generally use some counter-irritant to the skin, such as dilute linimentum crotonis, combined with a continuous compress, which

is applied warm and re-moistened night and morning. The stimulating liniment is applied as often as is necessary to maintain an eruption without causing dermatitis.

It must be remembered that pain in the knee-joint may be merely a reflex symptom of hip disease. I remember one case of an elderly man who had been treated in two hospitals for knee trouble and loss of power on walking. I was asked to see if anything could be done for him ; but I noticed that, as the man walked across the room, though the trouble was in his left knee, he supported himself by a stick held in the right hand. I at once diagnosed hip disease, as if the knee had been at fault he would have held his stick on the same side, whereas in hip cases the patient always holds it on the side opposite to the affected joint. This diagnostic point may be useful in such cases. In this case the man had never had the smallest pain in the hip, in which he had advanced atrophic disease, but very severe pain in the knee-joint, and attributed his loss of power in walking solely to the knee.

COMPLEX FACTORS IN JOINT DISEASE.

I have classified the various arthritic disorders in accordance with the types usually met with in everyday practice. It will be seen that each of these types is due to some definite physiological impairment, or some excess or deficiency in the normal constituents of the body. But we have to remember that the individual patient who may have one or other of these factors producing joint troubles, may have others peculiar to his conditions of life or his constitutional condition, or may have these factors in a greater or lesser degree, so

that the symptoms presented differ from those more commonly met with.

Thus, joint troubles due to depression of the central nervous system, which I have described under the name arthritis deformans, and which show no excess or diminution of lactic acid or phosphate of lime, are comparatively rare. But we meet with this neurosis in patients of the lactic acid and lithic diatheses. They have been exposed to severe shock or prolonged over-strain of the nervous system, and we see the results of this in the condition presented by the joints. In these cases the deformity is a factor superadded to the normal symptoms.

A patient with chronic rheumatism, chronic gout, or rheumatic gout may have a sound constitution and be of a robust type; but persons of weak constitution and who are debilitated may suffer from these disorders, and consequently the joint symptoms may have greater resemblance to those met with in rheumatoid arthritis, especially if there has been great wasting of muscle secondary to the disuse of the joints.

Such conditions, while they may make an accurate diagnosis more difficult to those who have not the opportunity of large experience, really give us a great amount of information apart from anything the patient tells us. The diagnosis is easier and more accurate if we do not start with the idea that we have to give a name to the disease, but only concern ourselves with what is actually wrong with the structures of the joint, and the condition of the excretory functions of the skin. We have to decide whether a deformity is due to failure of the muscular apparatus from disease or abnormal use of its function, or to some disorder of the central nervous system.

When we have made our minds clear on these points, we are in the best condition to consider the treatment of the individual case. Thus, although we may be impressed with the value of some particular dietary in patients who have lithates in their joints, we shall not at once put every patient on such diet, without regard to the fact that they may be too thin, or too fat, or whether they eat largely or too little. We may be impressed with the value of exercise in the same class of cases, but we should not encourage patients whose joints present neurosal symptoms to overfatigue themselves.

There are large numbers of arthritic patients who are best treated with abundant food and tonics, but there are many to whom the same treatment would do great harm.

We cannot differentiate these cases by the name we give to the disorder, but by the physiological conditions present in the individual patient.

The literature of this subject contains too much sweeping generalization, an effort to find a common cause, and therefore a common name, for symptoms which result from conditions often the reverse of one another, and which require recognition before efficient treatment can be prescribed.

The result of this is not only failure of the methods adopted, but the practitioner comes to regard such cases as lacking in interest and undesirable in the wards of a hospital. As a matter of fact, each case is an interesting problem demanding skill and judgement on the part of the physician. In acute disease he has always the *vis medicatrix naturæ* to help him in accomplishing his cure; in these cases, nature has failed, and successful results represent medical skill and add to the reputation of the profession.

CHAPTER XII.

'NEURITIS.'

During recent years the term neuritis has been used indiscriminately to represent pain in any part of the body, and therefore the word conveys no information concerning the tissues involved, nor does it give any indication for treatment. The word 'neuritis', in its literal sense, implies, of course, that the patient is suffering from inflammation of a nerve. This is a very rare affection, and does not come under my observation.

I propose to consider those cases which have been diagnosed as neuritis, and also some due to nerve disturbance but not recognized as such. They can be conveniently divided into two classes : (1) Those where the site of the pain is the site of the disorder ; (2) Those in which the site of the disorder does not correspond with the site of pain.

In the first class we may place rheumatism affecting the fibrous tissue of muscles, rheumatism of tendons, and lithates in the same, and rheumatism in the sheath of the nerve-trunks. In all of these tenderness is felt at the site of pain when the part is pressed upon. This is not in itself a diagnostic symptom ; but it enables us to locate by manipulation the particular tissue affected. Thus in muscular rheumatism the tenderness is diffuse and usually superficial. In rheumatism of tendons it is strictly localized to the tendon or tendons involved. In rheumatism of the sheaths of nerves it can only be found on deep pressure over the nerve-trunk, and the

area of tenderness is limited to the affected tissue. In all these cases pain can be elicited by any movement which puts the affected tissue on the stretch; in the case of muscular rheumatism by any effort which brings the muscles into action. It is the power to produce pain by stretching the tissue which distinguishes these affections from some others which I shall presently consider.

Muscular rheumatism is so easily diagnosed that it would seem impossible for any mistake to be made; yet the large majority of cases I have seen during recent years have been diagnosed as neuritis. One case which I saw many years ago impressed me very much with the necessity of physical examination before a diagnosis is given. The patient was a London physician who had suffered very intense pain in the back and limbs, for which he had failed to find relief. He had tried many forms of treatment, without result, and was worn out with pain and loss of sleep. On the morning I saw him he had written letters giving up his house and practice, as he felt that he would never be fit for work again. A brief examination showed that he had a severe attack of muscular rheumatism; in other words, the fibrous tissues of the muscles were loaded with lactic acid undergoing oxidation. I gave the opinion that he would be able to resume his practice within three weeks. He resented my diagnosis in spite of the favourable prognosis, and mentioned the names of three distinguished physicians who had diagnosed his case as neuritis. But neither of them had made any attempt at physical examination. I ordered daily pyretic treatment, and a fortnight later the patient resumed his practice; he wrote me some years later to say that he had had no return of the trouble.

Rheumatism of the tendons and fibrous tissues may be the result of a chill, but more frequently some strain upon the tissue is the direct cause. This is most likely to happen in persons who, not being accustomed to great muscular efforts, try to accomplish something greater than their capacity, or are forced to do so by some accident. The primary effect of the strain is to depress the physiological activity of the tissue; this causes lactic acid to accumulate, but during this period no symptoms are experienced. It is only when reaction occurs, which may be some time later, that pain develops.

Certain cases of sciatica are due to rheumatism affecting the sheath of the nerve, and these cases are most often due to sitting on a damp surface. A vehicle in which the cushioned seat has been exposed to rain, and then the surface has dried, so that the dampness is not recognized, is one of the most frequent causes of this painful ailment. It is readily diagnosed from the fact that if the extended limb is flexed upon the body, so as to stretch the nerve, acute pain is produced. Tenderness over the nerve-trunk is also found if deep pressure is made at a point mid-way between the great trochanter and the tuberosity of the ischium. None of these cases present any difficulty in diagnosis.

In the second class of cases, pain may be complained of in a limb, or at some part of the body; the cause of the pain or disability may not be readily understood, and the term neuritis applied to it.

If we take cases called neuritis of the arm, where very severe pain may be felt, we shall usually find it due to one of two conditions. The first is the decomposition of some small lithic deposit at the shoulder-joint: the region at or near the acromioclavicular

articulation is a frequent site. The patient is conscious only of the pain radiating down the arm, and is not aware of a point of acute tenderness in the shoulder which may frequently be covered by the finger-tip. In these cases a branch, probably, of the circumflex nerve, crosses the area of inflammation and sets up irritation in the main nerve-trunk, which makes itself evident, not in the nerve-trunk itself, but at the area of its distribution. Pain is frequently felt at the point of the insertion of the deltoid muscle, and before the advent of the word 'neuritis' this pain was frequently called deltoid neuralgia.

Now it is obvious in these cases that treatment administered to the site of pain can accomplish no good results, while, if we treat the point of irritation at the joint by soothing and anodyne applications, excellent results are obtained. After the acute symptoms have passed, I find it a good plan to paint the part where the lithitis exists with liniment of iodine, allow it to dry, and then cover it with zinc plaster. In severe cases, where there is some effusion, I use a blister, which gives relief.

In these cases, although the pain is excited by a subacute inflammatory process, there is no inflammation of the nerves which cause the pain, so that the word 'neuritis' is not applicable: 'lithitis' or 'gout' is a more accurate diagnosis.

But we meet with cases of neuritis in the arm in which the lithate plays no part. There is pain radiating down the arm which may extend to the fingers; it may be more or less severe, and have a paroxysmal character. The examination of such a case is never complete unless we place the finger immediately above the upper border

of the clavicle and press deeply, and then carry it along the bone so that the nerve-trunks entering into the brachial plexus are pressed between the finger and the tissues beneath. One or more points of great tenderness may be revealed by this proceeding, although the patient is wholly unconscious of any trouble in this region. If, without relaxing the pressure, we move the finger to and fro so as to cause friction transversely to the nerve-trunk, we shall excite tingling down the arm extending to the finger, and, if we are too vigorous in our efforts, acute pain will be produced, or an aggravation of the pain already existing. If we are more gentle in our friction, and continue it for a few minutes, the pain caused by the operation gradually diminishes, and, if the patient is in pain at the moment, the friction gives relief. If we now continue the process until there is a feeling of warmth under the finger owing to increased circulation, which may take from seven to ten minutes, the pain in the arm is relieved, and the part to which the friction has been given is very much less tender to the touch, or the tenderness may altogether disappear, whilst the increased circulation remains. By this simple proceeding we are able to demonstrate the nature of one of the most important causes of the symptoms called neuritis, and which may appear in any part of the body.

There is an asthenic condition of the nerve-trunk, due to diminished blood-supply. This condition produces no symptoms at the site of disorder, but causes pain or loss of nerve power at the point of distribution of the affected nerve. We know that the condition is one of defective blood-supply to the nerve, because, as we increase the circulation, the pain in the area of distribution, and the tenderness over the nerve-trunk,

diminish, or are temporarily arrested. It is obvious that if we start by diagnosing this condition as neuritis, and regard these symptoms as indicating an inflammation of the nerve, we shall resort to treatment the opposite to that which is beneficial, and finally curative, and we shall also apply our remedies to the area of pain instead of to the site of the disorder. In other words, the diagnosis 'neuritis' contra-indicates the necessary and curative treatment.

This asthenia of the nerve-trunks is one of the most frequent disorders which come under my observation. I do not think there is a single nerve-trunk in the spine which I have not seen affected in this manner. The symptoms produced are distant from the site of trouble, and if the cause is not recognized they are difficult to account for, and may lead, and have led, to errors in diagnosis of a serious character. The diagnosis of neuritis is not the worst thing that can happen to a patient with this condition.

The cause of these symptoms is unquestionably traumatic. When the brachial plexus is involved, it is a consequence of some act of exertion in which the nerve-trunks of the brachial plexus are unduly stretched. We know, when a ligament is sprained, that we first of all have acute pain and some effusion. When this has subsided and the use of the joint is restored, the site of the injury may remain in an asthenic condition for many months, although the patient may be unconscious of the defect. Under these conditions lactic acid may collect in the affected tissues, and finally there is a reaction, and rheumatic symptoms may appear. Or, in the patient with the lithic diathesis, there may be failure to dissolve the dead cells of the tissues, and lithates may form. All this may take place and

present no symptoms for months or years after the accident.

In overstrain of a tendon from some act of exertion, the symptoms at the time are different. There is not, of necessity, any pain at the time of strain, or the symptoms may be so slight as not to excite attention or remain in the memory. But the physiological consequences are precisely the same as in the sprained ligament. The tendon may become the site of rheumatic trouble, or lithates may be deposited.

In regard to sprain of the sheath of a nerve-trunk, there may be pain at the moment, or even for a few days later, but it is usually transient. This pain may be felt at the site of injury. It may not be for many months after the injury that symptoms begin to appear in the area of distribution of the nerve, less frequently at the site of injury.

The asthenic condition which occurs in ligaments or tendons has a natural tendency to recovery, because both are brought into functional activity during the movements of the body. This cause of natural cure is absent in the trunks of nerves. They are like so many insulated wires passing through the tissues; the sheaths of the nerves take no part in their functional activities, and receive no stimulus from them. As a result, injuries to the sheaths of nerve-trunks have no natural tendency to recovery. This being so, we might expect the symptoms to continue unabated through life, but this is not the case; the patient may have attacks of pain or disability due to loss of function in the nerve, and this may last for an indefinite period. Then there may be periods during which the patient has no symptoms, the trouble is apparently cured, but from time to time they recur,

and this recurrence is always in the same area as the first attack. If, however, at any time after the injury, whether the patient has symptoms or not, we press over the affected nerve, we shall find a point of acute tenderness, showing that the injury remains although no symptoms may be present.

In all cases of pain in any part of the limb, thorax, or abdomen, when there is not an obvious cause for it, the back should be examined.

In the cervical and dorsal regions, a line may be taken about two inches from the spines of the vertebræ on either side. The finger is pressed firmly, and a to-and-fro movement made, transversely to the nerve, as the finger is carried down the spine. This will only occupy a few minutes. The brachial plexus is best examined immediately above the clavicle in the manner described, or friction may be made over the nerves in the axilla.

The lumbar plexus, which is more subject to traumatism than any other nerve-trunks, will show an area of tenderness, if we take a line from the crest of the ilium to the vertebral column, and press about two inches below the centre of this line. The sacral plexus is easily examined by deep friction, about two and half inches from the centre of the bone on either side.

Injuries of the lumbar plexus occur with greater frequency than those of other nerve trunks because it is apt to be sprained by twisting the body in an effort to save a fall. In many cases, where the patient has a fall, and may be severely bruised, the local injury is of far less consequence than the injury to the nerve-trunk, which may give no evidence of its existence for months or years later. Both lumbar plexi may be sprained by the attempt to lift a heavy weight with the

body in a flexed position. Thus, leaning across a bed and trying to lift a patient may cause this injury.

The symptoms produced by these injuries, when they appear, will be pain in the area of distribution of the nerve affected, and it is a diagnostic symptom in these cases that the nerve which conducts the pain is tender on pressure. Thus, when the lumbar plexus on one side is injured, both the external and internal cutaneous nerves of the thigh are tender to pressure, or to friction over them. The best point to examine the external cutaneous nerve is at a point mid-way between the great trochanter and the external condyle of the femur. The internal cutaneous nerve is most superficial at a point about four inches above the internal condyle of the femur, on the inner side of the thigh.

Some years ago the statement was made that it was impossible to distinguish between the symptoms of stone in the kidney and lumbar neuralgia. If by lumbar neuralgia we mean the class of case I am describing, the diagnostic symptom is that in this affection the internal or external cutaneous nerves, or both, will be tender to the touch ; in pain caused by stone in the kidney this does not happen.

It is less common to have pain at the site of injury in cases of traumatism to the nerve-trunks. There can be little doubt that lactic acid accumulates in the fibrous tissues of the sheath of the nerve, and by its presence may cause thickening, and thus interfere with the nutrition of the nerve. I called attention to the fact that the thickened cell-wall in chronic rheumatism was one of the causes of the wasting of muscles and the tissues, and some interference with the nutrition of the nerve must occur if the walls of its sheath are thickened from the same cause. Except in the case of

the sciatic nerve, which is stretched in certain movements of the body, there is nothing to excite reaction in the nerve-trunk. The thickening of the sheath with its local tenderness may remain for twenty or thirty years without any local pain being exhibited in the nerve-trunk, although pain in its area of distribution may be frequent or recurrent.

I have met with some exceptions. One was that of a lady resident in a distant colony who was seized with most acute pain in the lumbar region on both sides and involving the hips. The pain was so acute that the patient had to be kept under the influence of morphia. When she had sufficiently recovered she was sent to London for examination, as the symptoms were obscure. She saw one of our greatest and most experienced surgeons, who, having given an hour to examining her, felt uncertain about the cause of the pain and disability, and asked a very distinguished authority on diseases of the nervous system to see her with him. They finally pronounced it a case of acute arthritis of both hips. I subsequently saw the patient and found that she had sprained the lumbar plexus on both sides, and that this had caused severe pain both at the site of injury, as well as in the area of distribution. I suggested that she had tried to lift a weight with the body flexed at some time before the symptoms appeared. She strongly insisted that this could not be the case, as she was too old for any laborious employment. The next day her daughter called to tell me that her mother had since recollected that during her husband's illness she had tried to lift him in bed, and felt a severe pain in her back ; that the pain disappeared in a few days, and she had forgotten it. It was four or five months after this that the severe attack occurred.

I give these particulars because in many cases the patient will deny the history of an accident, as, owing to the lapse of time between the symptoms and the cause, no impression has been left on the mind.

While in the majority of cases of injury to the lumbar plexus there is pain as the chief symptom, there are some in which it is absent and the motor fibres of the nerve appear to be alone affected. A common symptom is that the knee gives way whilst walking, and the patient falls down in consequence. This is due to a lack of nerve power in the internal and external cutaneous nerves, which supply the structures responsible for supporting the joint. The symptoms, both of pain and loss of power, depend upon diminution of the normal functional activity of the nerve, so that if anything occurs to depress the general nervous system it will make itself evident in the weakest nerve. Thus a patient with injury to the lumbar plexus will walk quite well and continue to do so, but one day, from over-fatigue or some cause of nerve depression, the knee will suddenly give way and the patient will fall down. This symptom does not usually occur in patients who suffer pain from lumbar-plexus injury, and those who have the symptom very seldom complain of pain, which shows that the sensory fibres are depressed in one case and the motor in the other.

As I have stated, the symptoms do not usually appear until a considerable time after the accident ; but I had one case in which it appeared in the first twenty-four hours. An elderly lady, whilst walking, came to a small step which she did not notice, and in consequence fell to the ground. She walked home and felt none the worse for the accident ; but the next morning, on

rising from bed, her knee gave way and she fell to the ground. The symptom persisted, and two surgeons had been consulted before I saw her, and could find no sign of injury. But when I touched the point I have mentioned, the pain was very acute, and friction over the point eventually cured the trouble.

Injuries to the lumbar plexus may cause spasm of the muscles of the hip so as to produce a shortening of the limb on the affected side. One case of this kind came under my notice many years ago. The patient was a lady 27 years of age. She complained of severe neuralgia, and came to me for treatment of this trouble. On examination I found that she had a pronounced lateral curvature of the spine, and one leg was four and a half inches shorter than the other. She was wearing steel supports for the spine, a steel-support from the ankle to the knee, and a high boot.

I formed the conclusion that the original condition had been produced by spasm of muscle due to injury of the lumbar plexus, and that efforts to remedy this by mechanical means had pushed up the hip and caused the condition of the spine. I kept the patient in bed for a fortnight, and ordered cold sponging to the whole spine (which was irritable to percussion), followed by deep effleurage until an active circulation was produced; also friction over the lumbar plexus on the affected side. I then put traction on the shortened limb by means of a weight and pulley. At the end of the sixth week the scoliosis and the shortening of the limb had disappeared; the patient was able to walk without any supports, and quickly resumed active life. She had been for some years under the treatment of an orthopædic surgeon, who did not recognize that by progressively increasing the height of the boot

he was simply tilting the pelvis and distorting the spine. There was no real shortening of the leg from the first.

Recently I had two cases, at the same time, under treatment where injury of the lumbar plexus had produced spasm of the muscles of the hip, and shortening of the limb as a consequence. One was an ex-cavalry soldier, who had so much swelling and pain in the region of the hip, combined with a shortening of over three inches, that at the hospital from which he was sent to me, the diagnosis of arthritis of the hip-joint was made. Curiously enough, it was apparently confirmed by an *x*-ray photograph. The man had all the symptoms of an advanced case of arthritis of the hip, but the disorder had only existed for a few months. He had been examined for tuberculosis with negative results.

In this case there was not only acute tenderness over the lumbar plexus, but pressure produced spasm of the muscles. There was acute tenderness over the external and internal cutaneous nerves of the thigh. Now in cases of hip-joint disease we may have pain reflected by these nerves, and felt both in the thigh and the knee-joint, but the nerves themselves are not tender to the touch. I at first thought that this injury was due to one of the many falls he had had from his horse, and reported this opinion; but subsequent examination showed that both lumbar plexi had been sprained, and this must have happened from lifting a heavy weight. On inquiry, I found that after leaving the army he had been in attendance upon a very heavy man who was an invalid, and when leaning over the bed and trying to lift him the injury had taken place. In this case vibration over the affected nerve, and traction by weight and

pulley at a later stage of treatment, quite removed all the symptoms, and the man was restored to work.

The other case was that of an elderly man who had sprained the left lumbar plexus during a fall. The condition was much less severe, and the limb of the affected side which was shortened by two inches became normal by simple rest in bed and treatment of the affected nerves.

Injuries to the sacral plexus are caused by a fall in the sitting posture. They are far less frequent than injuries of the lumbar plexus, not because such falls are uncommon, but because it requires a severe fall to produce the injury. One severe case, involving the nerves on both sides, was due to a lady intending to sit on the box seat of a coach, missing the seat, and falling to the ground in the sitting posture.

The pain in these cases follows the course of the sciatic nerve, and this nerve is tender to the touch. This is also the case in rheumatism of the sheath of the sciatic nerve, but in the latter case pain can be produced by stretching the nerve, as when the extended leg is flexed upon the body. This symptom does not occur when the pain is due to injury of the sacral plexus. In a local rheumatism of the sheath of the nerve the pain is aggravated by walking; but this symptom is not so prominent in injury of the sacral plexus. The pain is apt to occur when at rest, and also in bed; and especially after fatigue. There is tenderness on pressure over the sacrum at the points where the nerves emerge which is not present in rheumatism of the sheath of the nerve; nor is it in cases of pure neuralgic sciatica.

These cases have no tendency to natural recovery, and the baths which would benefit rheumatism of the

sheath of the nerve may quite fail to relieve the condition. But when the true nature of the disorder is diagnosed, the fact that the nerves are placed more superficially than those of the lumbar plexus makes them more easy to treat by physical methods, and rapid improvement takes place.

These cases are very interesting, because the symptoms indicate the nerve-trunk affected, and the possible causes of injury to special nerve-trunks are limited, so it is not difficult to describe an accident which the patient has met with and wholly forgotten. In one case there were tender points over the cervical and dorsal nerves, and also over the left lumbar plexus. This could only have been caused by the patient falling downstairs on her back and then something occurring to twist the body to one side. This diagnosis was confirmed by the patient. She was on board ship, twenty years before, and had gone on to the upper deck; as she was descending the companion ladder her foot slipped and she came down the steps on her back, and, just before she reached the bottom, someone trod on her dress and she twisted over and reached the deck on her side.

I have seen many cases where the patient was liable to attacks of acute pain of rather an alarming character in the region of the heart. In such cases I frequently find a tender point over the 3rd or 4th dorsal nerve, and by passing the finger along the lower border of the ribs the tender nerve can be traced to the site of pain. Friction over the affected nerve will give relief during an acute attack, and if the nerve is treated the return of this trouble may be avoided.

In all obscure pains over the stomach or liver which come on periodically and which are not easily accounted

for by any gastric or hepatic cause, the region of the 6th and 7th dorsal nerves should always be examined, as it may solve the whole difficulty. I had one case in which the 8th and 9th dorsal nerves on the right side were affected, causing very acute and continuous pain. The nerves had been strained whilst playing polo. The patient, a naval officer, was quite relieved by treatment. Five years later he had a return of the pain, and as a consequence abscess of the liver was suspected and he narrowly escaped operation.

Some cases, regarded as recurrent appendicitis, are due to injury of the 11th and 12th dorsal nerves on the right side. Acute tenderness over these nerves will be found. If this condition is discovered during an acute attack, and friction is given to them, pain will be relieved in about ten minutes. To prevent return of the trouble, friction should be given daily until the nerves are no longer tender to the touch.

A further diagnostic method in all painful affections of the abdomen due to nerve injury is to pinch up the abdominal wall over the site of pain, and hold it firmly between the thumb and fingers of one hand, while pressure and a rolling movement is made to the tissues with the other hand. This means that the abdominal contents beneath the abdominal wall are in no way affected by the manipulation. If, then, pain is produced, we know that it is due to the nerves of the abdominal wall, and not to any inflammatory condition of the intestine. In the cases I have described, the abdominal wall will always be found tender over the site of pain.

I have already mentioned that patients with the lithic diathesis are more liable than others to recurrent pain in the gall-duct, due to thickened bile or concretions,

and this pain is frequently reflected to the region of the appendix. Therefore, in cases of suspected appendicitis, the region of the gall-bladder should always be examined in addition to lower dorsal nerves.

In one lady who had undergone very strenuous and unaccustomed exertions during the war, I found acute tenderness over the 8th and 12th dorsal nerves, and also over the lumbar plexus, all on the right side. On account of pain, the right ovary and the appendix had been removed; but she was told that they were free from disease. A further operation was proposed for 'floating kidney'. The abdominal muscles were quite firm, and there was nothing to indicate any displacement of the kidney. Pain in the thighs and loss of power in the knee-joint were attributed to this displacement of the kidney, but such displacement would not cause the tenderness and asthenic condition of the internal and external nerves of the thigh which existed in her case. This is another diagnostic symptom, because reflex pains are not accompanied by tenderness over the trunks of the conducting nerves.

These facts led me to examine the corresponding nerves on the left side of patients who had chronic colitis, or who were subject to repeated attacks. In a large number of cases I have found tenderness over the 11th and 12th dorsal nerves of the left side in these cases, and also I have found treatment of these nerves, when tender, produce better results than any other method. Previously I had formed the conclusion that colitis always represented an asthenic condition of the nervous system and that no local treatment was of value unless the general health was restored. I think this view is correct, but it is obvious that if we can find a definite cause for the local asthenia, and remove it,

the results of treatment will be much more rapid and complete.

The cause of injury to these nerves in the cases I have met has been usually the effort to lift some heavy piece of furniture. Thus, a young lady tried to move a piano, and was at last obliged to desist because the strain was too great. Her sister then made an effort to do the same thing, but was also obliged to give up. Both these ladies developed colitis about the same time, a few months later.

I recently had a man under treatment who was subject to severe pain in the region of the descending colon, and also to great distention of this part of the intestine. He had treatment for many years, and adopted various dietetic measures without relief. I found marked tenderness over the 11th and 12th dorsal nerves of the left side. I found that he was the manager of a large piano business, and he admitted that he had sometimes helped to move a piano, but had not attempted it since he had this trouble. Treatment of these nerves made a rapid and permanent cure of his condition.

Efforts to lift a weight when the spine is flexed are likely to strain the lumbar plexus ; but the same efforts, when the body is erect and close to the object to be lifted, appear to put the greatest strain on the lower dorsal nerves.

I have seen other cases where injury to nerve-trunks, and especially the lumbar plexus, has been followed by loss of motor power, particularly during fatigue or in depressed conditions of the nervous system, which has led to the diagnosis of sclerosis, and unnecessary alarm caused to the patients and their friends.

These facts point to the necessity of examining the

condition of the spinal nerves in all cases where pain and disability exist. The electrical methods adopted by neurologists are not adapted to the discovery of these lesions. Simple pressure and friction with the finger is all that is necessary in order to detect them.

The same is true in regard to the treatment of these disorders. We might expect that having to deal with an affection of the nerve, we should find electricity our best resource. The high-frequency current will often relieve pain, and the galvanic current improve the nutrition of the nerve, but neither of these will take the place of mechanical friction with the finger. It requires to be commenced very gently, the movement being always transverse to the nerve, and the pressure gradually increased as the functional activity of the nerve becomes greater and the tenderness consequently less. It needs to be continued until a feeling of warmth is produced in the part, and with it a subsidence of the pain and tenderness. This treatment usually has to be continued daily for three weeks or a month in order to secure curative results.

There are some cases in which the tenderness is so acute that the treatment would be exceedingly painful, and as a routine measure it is desirable to commence the treatment by electric vibration over the affected nerve. If the vibrator has a rotary action of the applicator which is checked when applied to the body, the energy is conveyed to the tissues and a rise of temperature takes place. This does not happen when the rotation of an excentric wheel simply causes a percussive motion to the applicator. After vibration has raised the temperature of the part, friction to the nerve becomes painless, and can be done much more vigorously than when it is not used.

While there are some cases where the treatment will succeed without other measures, there are others where the nerve-trunk is so saturated with lactic acid that some form of pyretic treatment should be given at the same time. In fact, the best results are always attained in this way.

The immediate effect of treatment is always relief of pain, and this proves that it is indicated; but no case can be regarded as cured until the nerve is no longer tender to the touch. Thus relief of all the symptoms is not of necessity a cure. While the nerve-trunk remains tender, there is always liability to recurrence.

The term 'neuritis' cannot be properly applied to any of the cases I have described. Pain due to an asthenic state of a nerve, this condition being due to a mechanical cause, the result of injury, might be called traumalgia. This would distinguish it from a neuralgia, which might be cured by medicinal treatment. Traumalgia necessitates local treatment to the site of injury. The same is true of those cases when the motor fibres of the nerve are alone affected, and the symptoms are those of loss of power at the point of distribution. Such cases might be described as traumasthenia of the particular nerve affected. In both cases we have to remember that the cause of the continued asthenia of the nerve is the thickening of its sheath, and that we have to adopt some means by which the additive lactic acid, which causes the pressure, is removed.

CHAPTER XIII.

THE NURSE'S PART IN TREATMENT.

In the chapter on Pyretic Treatment I have described some of the simple methods by which the conditions causing the 'rheumatic' attack can be averted, and which may also be used with advantage when acute symptoms have occurred. But when the changes in the tissues have reached an advanced stage and the joints have become crippled, systematic treatment is necessary, and this needs efficient appliances and also the aid of nurses specially trained for this work. At the Lansdown Hospital and Nursing Home at Bath I have had a long experience in the instruction of nurses in carrying out treatment, and some hints on the points special to these methods may be useful to the practitioner.

In the first place it is essential that the nurse should clearly understand the physiological effect aimed at in any treatment she has to administer. For this reason it is desirable that they should be selected from the educated classes.

They must learn that the thermometer is not an instrument solely used for detecting the presence of fever; but that it is the most valuable means we possess of estimating the activity of the chemical processes taking place in the body.

Unless it is made an absolute rule that the register of the thermometer be shaken down to 95° F. before the temperature is taken, the subnormal temperature

will not be recorded. A person whose temperature is always subnormal, even if he be otherwise in good health, has that condition of imperfect metabolism which may lead to arthritic developments. If the morning temperature suddenly falls during treatment, it most usually indicates over-fatigue on the previous day. If it persists in spite of rest, it may indicate that the treatment itself is the cause of fatigue and must be modified. In such cases the galvanic bath on alternate days with the thermal couch (or pyretic bath) is usually sufficient to correct the difficulty.

It will be noticed that few patients with the rheumatic or lithic diatheses have a morning temperature of 98·4° F. unless a febrile condition is present. By the words 'normal temperature' we mean the temperature normal to the patient, and not the theoretic normal.

Another point of importance is the difference between the morning and evening temperature. A wide variation in the absence of any febrile condition usually indicates some debility; if it is both wide and erratic, it points to fatigue of the nervous system.

It is usual with patients to show a greater variation at the commencement of treatment than as it continues, and when it becomes very slight indeed we can be satisfied that the patient's nervous equilibrium has been restored. It will be seen, therefore, that the temperature chart of a patient whose temperature never exceeds the 'normal' line is very instructive, and helps us in regulating not only treatment but the daily life of the patient both as regards exercise and nervous excitement.

Thus one patient whose temperature was subnormal suddenly had a febrile rise every night, and I failed to

account for it. On close examination I found that she played bridge every afternoon ; I stopped this, and the temperature became normal.

When the temperature is above normal it is important to consider it in relation to the rate of the pulse. As a rule we may regard a rise of temperature without any great acceleration of the pulse as a simple hyperthermia, showing active chemical change of a beneficial character.

A rapid pulse with a moderate temperature is very uncommon in such cases, but when it occurs we may suspect a large amount of *free* lactic acid is irritating the vascular system. In such cases the skin, if functionally active, shows a very acid reaction. The free use of some simple alkali, such as bicarbonate of soda, may then prove very useful and control the symptoms ; when it does so, we know that our diagnosis is correct.

In all cases of continued fever, during acute attacks, it is very important to observe the skin reaction in relation to the temperature. If the latter falls and the symptoms abate while the skin reaction shows marked acidity, it is an indication to continue pyretic treatment, as otherwise we shall have prolonged convalescence and liability to relapse. I have already called attention to this point, but it is so important that I make no apology for repeating it.

I have previously described the construction of the 'thermal couch' which I have found the most efficient means of giving pyretic treatment. The patient is in the horizontal position and surrounded with moist warm air (instead of vapour or steam), and the temperature can be exactly regulated during the course of the bath, which is an important factor. This is done

not only by altering the temperature of the boiler, but by controlling a number of small electric lights placed in the cover of the couch.

The duration of the treatment is determined by the moment when sweat appears upon the forehead of the patient. If this should occur too rapidly, as it may do in patients of the lactic-acid diathesis when the skin is active, it is an indication that the ordinary temperature of the bath (105° F.) is too high, and it is reduced at the next bath. If, on the other hand, no sweat appears upon the forehead in twenty to twenty-five minutes, as may happen during the first days of treatment of patients with suppression of the cutaneous function, the bath is discontinued and repeated the next day with an increased amount of moisture in the air of the bath. It is very seldom that cases occur where the normal functions of the skin are not restored within three days. Twelve days is the longest period I have known before the skin resumed its functions.*

When the skin acts very freely it is often a good practice to turn off the heat ten minutes after the patient has entered the bath. Directly indications are given that the skin is acting freely, the temperature of the patient is taken. While this is being done the reactions of the skin are tested. As a routine method that of the forehead, chest, palms of the hand, and soles of the feet are observed and recorded.

The temperature before and after the treatment, together with the four skin reactions, being recorded

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* Nurses should be specially warned against attempts to increase the efficiency of the treatment by raising the temperature of the bath. Its object is to restore and stimulate a normal function, not to obtain sweating by exhausting the cutaneous nerves, the secondary effect of which is to diminish their functional activity.

each day, make a chart which gives exact information concerning the patient's progress. Without these observations one has no knowledge of the actual conditions, and no guide to the number of treatments required. It is as a result of the information thus obtained that one realizes the large number of baths which are necessary to free the tissues from the excess of lactic acid in many cases.

At intervals during the treatment specimens of the sweat are taken for microscopical and chemical examination. I find the most convenient method is to use an ordinary teaspoon, which is passed over the surface of the skin, and the contents are then poured into a small bottle, labelled with the name of the patient. Some distilled water from the vapour of the bath is mixed with the sweat, but this is of no consequence. The chemical examination is of importance, especially in cases where a neutral or faintly acid reaction persists. It will be frequently found that lactic acid exists in large quantities but is neutralized by ammonia. In a large number of acid sweats, ammonia will be found.

It is easier to train nurses to give baths efficiently than it is to make them successful manipulators of crippled joints. It does not follow that because a nurse has had a course of training as a masseuse she will secure good results in these cases, although the manual dexterity she may have gained is an useful factor. The point of chief importance is to train nurses to recognize the degree of pressure they are using when they handle a patient. The skin pressed upon the subcutaneous tissues is the first degree, the skin and tissues pressed upon the muscles is the second degree, and deep pressure of all the tissues on the bone is the third degree.

Having secured the correct pressure in any particular condition, they must be taught to maintain it while moving the hand either laterally or in a circular direction, and, while the movement continues, change from one degree of pressure to another. They will thus obtain the muscular sense that is necessary to all manipulative treatment. The second most important point is that while pressure may vary, contact must be always maintained.

The 'pétrissage' movement so much favoured by the massage school is very objectionable unless it is very well done. The staccato pinching of the muscles is irritating to the cutaneous nerves. Pressure of the third degree with the whole hand and thumb, alternating with pressure of the second degree and passing gradually into one another, while a slight rotatory movement of the tissues is made as the hand glides along the limb, is more efficient and is soothing.

But in the class of cases with which we have to deal, resisted movements of the joints are much more frequently called for than massage manipulation. There are two points which it is necessary to impress upon nurses in giving these movements. Each movement must be complete, the flexion must be carried as far as is possible without causing unnecessary pain, and the same is true of the extension movement. When skill is obtained in the treatment of contracted joints, the movement commences with resistance and ends with the act of assistance, the greatest care being taken not to carry the assisted movement too far. We can only obtain the help of the patient, which is the most valuable part of the movement, while he has the most complete confidence in the manipulator.

The second point is that the nurse must be instructed

to watch the patient carefully and see that the effort he makes is only the result of the contraction of the muscles proper to the movement. I have seen patients quite exhausted after a few flexion and extension movements of the limb, because at each effort they have contracted a large number of muscles, not concerned in the movement.

All resisted exercises are best ordered by numbers, i.e., 3, 6, 8, or 10 to each joint. In no case should movements be continued if fatigue is produced. Quite apart from contracted joints, many rheumatic patients have defective expansion of the chest, and when there is free movement of the shoulder-joints, chest-expansion exercises are of great benefit. The arms of the patient are raised to their fullest extent, and then the patient is desired to draw them down until the elbows touch the sides, while the nurse resists the movement.

Personally, I order the patient to make a deep inspiration as the arms are raised, and to hold the breath during the resisted movement.

I know that the massage school is opposed to the patient holding the breath, but I judge by the results produced. We naturally hold the breath whilst yawning, which is an involuntary effort to stimulate the heart. The pulse is always taken before such movements, and seldom more than six are given, when the pulse is again taken. It will be found that the beat has increased in volume, and become slower, as a result of the treatment. If it should become quicker it shows that the movement has not been properly given; therefore it is necessary to have the pulse taken before and after as a routine measure. The cause of failure is usually that the movements have been performed too quickly—at least four normal respirations should take place between

each movement. A single inch of increase in the circumference of the chest represents an enormous addition to the intake of oxygen into the lungs during the twenty-four hours, so the importance of increasing the chest capacity in all cases where there is defective oxidation, cannot be overestimated. Nurses vary very much in the results they obtain from physical treatment, and as a rule I find that sympathy is a more valuable asset to such nurses than strong muscles.

The methods I have described have been the means of restoring a large number of crippled patients to a useful life; but I do not forget that these results are due to the patient labour of nurses who, in doing their work, are following one of the higher and most useful branches of their profession.

One good reason for the treatment of such patients in the wards of general hospitals is that the training which renders nurses efficient in this treatment fits them for all the other duties of nursing. It trains their observation, teaches them to control exactly their muscular activities, and gives them a sense of responsibility and a pride in their accomplishment when the task is completed.

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